

Obstetrics and Gynaecology

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Name: _____

Subject: _____

Obstetrics and Gynaecology



25/4/18

OBS & GYN/AE

①

RELEVANT ANATOMY

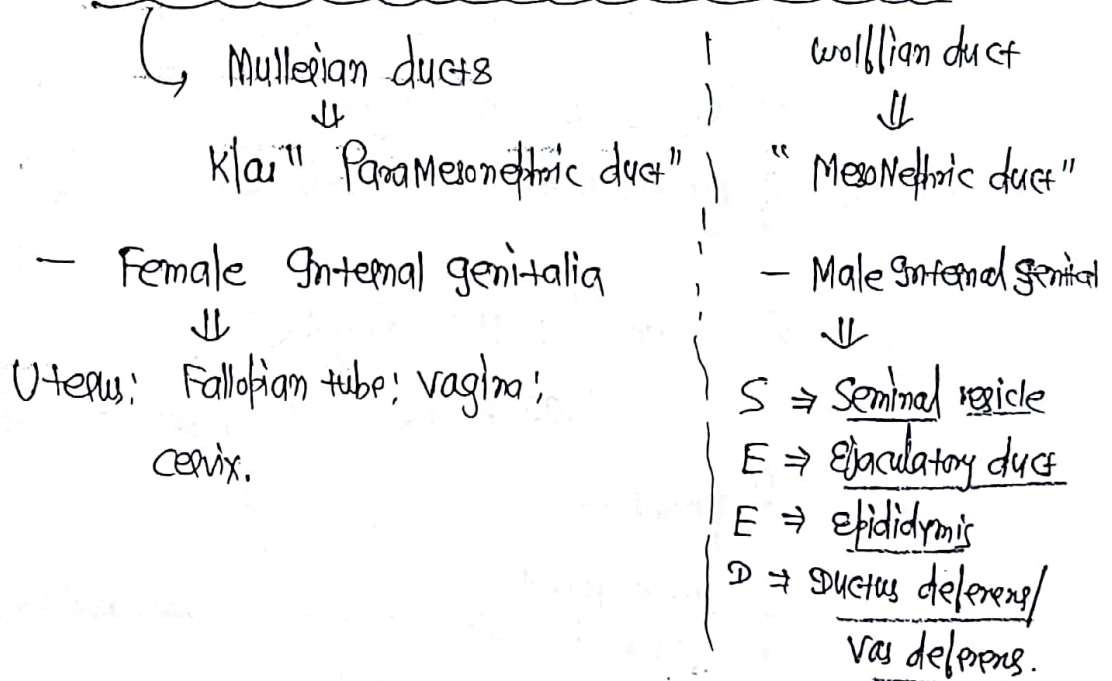
GONADS

Internal Genitalia

External Genitalia

Internal Genitalia \Rightarrow Uterus; cervix; Fallopian tube; vagina

embryological structure from which it develops \Rightarrow



* Both the ducts appear in every foetus @ 6 weeks.

* Müllerian duct is lateral to Wolffian duct,
 \downarrow either σ or f

* Müllerian disappears @ 9 weeks / Wolffian disappears in f .

Mullerian ducts

Disappear \Rightarrow In Male

Why??

Testis

MIS (Mullerian Inhibiting Substance)

\hookrightarrow * g/silateral

\rightarrow Released by "Sertoli cells"

\hookrightarrow Start producing
@ 7 weeks

- Mullerian duct will persist
In female b/c of Absence
of MIS

* Estrogen is Not Required for development of uterus,
vagina, Cx etc; for it absence of testosterone
Required.

\hookrightarrow Medial part of broad Ligament
Paroophoron
Epoophoron
Gartner's duct
Hydatid of Morgagni
Klay's organ of Rosen Muller's
all all contents
of broad Ligament

Wolffian duct

In Female

Why? b/c absence of testosterone

- Wolffian duct is persist In Male child

\Downarrow
Testes

\downarrow
Testosterone \rightarrow Leydig cells
 \Downarrow @ 8 weeks

cause development of Wolffian duct

* Remnants of Wolffian duct
In Female

- i) Epoophoron - cranial Remnants of Mesonephric tubules
- ii) Paroophoron - caudal Remnants of Mesonephric tubules
- iii) Gartner's duct - caudal Remnants of Mesonephric ducts

* Gartner's duct Sometimes pr. as cyst in the vagina

* Gartner's cyst \Rightarrow In Antero Lateral wall of vagina ⁽²⁾

9/9 \downarrow B Bartholin's cyst \Rightarrow In Posterior Lateral wall of vagina

M/c cyst of vagina/vulva \Rightarrow Inclusion cyst

\downarrow
Located @ Lateral wall.

* Remnant of Mullerian duct in Males \Rightarrow

while (Appendix of testis = klas. "Hydatid of Morgagni".

Appendix of epididymis is Remnant of Wolffian duct. ^{1P}

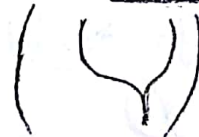
* Mullerian duct \Rightarrow 2 in No.

\downarrow both ducts fuse & form uterus in 10 weeks

We can differentiate internal genitalia @ 10 weeks

direction of Fusion — Fusion begins from centre then it moves cranially/caudally.

direction is from caudal to cranial.



* Initially Uterus is solid organ; Later cavity formation occurs @ 18-20 weeks.

by dissolution of a midline fibrous septum

Potential cavity

↳ to accommodate something

* if Fundus is convex upwards \Rightarrow No fusion Abnormality

if Fundus is dipping \Rightarrow Fusion Abnormality seen.

* Complete septate defect \Rightarrow



* Incomplete septate defect \Rightarrow



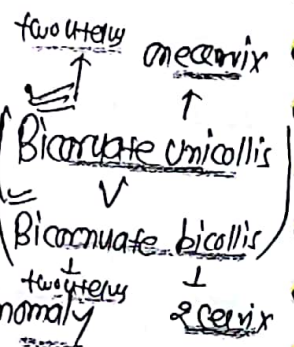
* M/C Mullerian Anomaly \Rightarrow Septate Anomaly

* 2nd M/C Mullerian Anomaly \Rightarrow Bicornuate uterus

* M/C Mullerian Anomaly also abortion

* M/C Mullerian Anomaly also infertility

* Worst Reproductive outcome



* In Diadelphous uterus both Mullerian duct form Anomaly (complete Lack of Fusion)



↳ complete failure of Fusion

vaginal septum

* Müllerian Anomaly = vaginal septum \Rightarrow Didelphus (3)

* Bicornuate Uterus has good Reproductive outcome

\hookrightarrow What pregnancy complication: \hookrightarrow Pre-term Labour
Most Likely to do Abortion

* Didelphus Uterus also has good Reproductive outcome

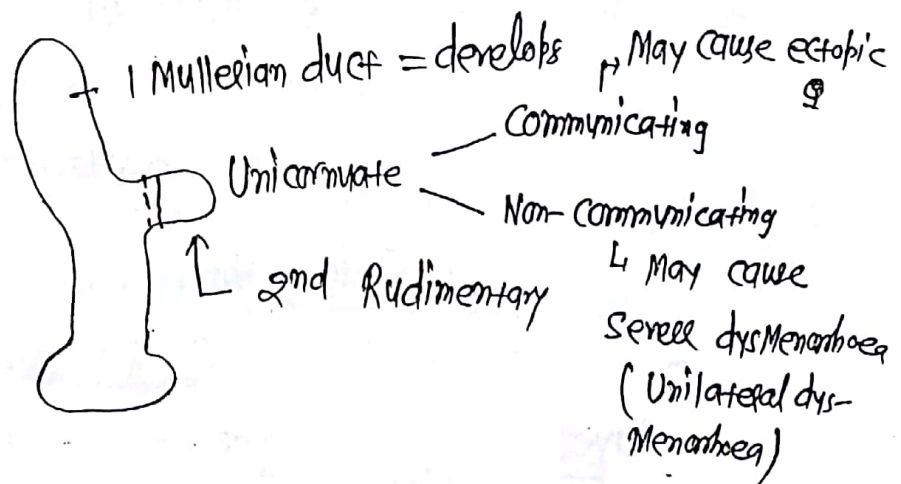
\hookrightarrow What pregnancy complication \hookrightarrow Pre-term Labour
Most Likely to do

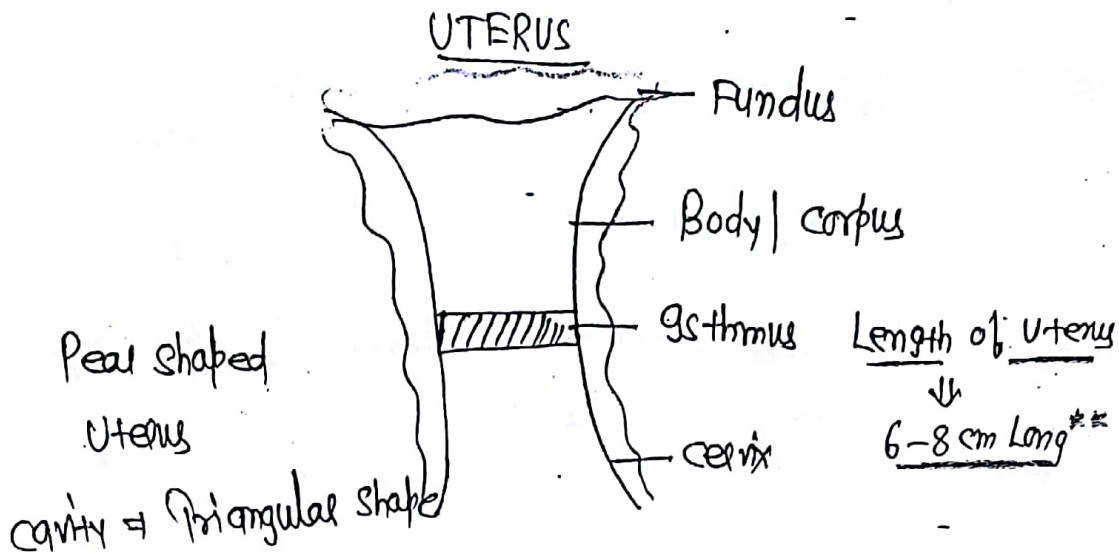
* Usually Corrective Surgery is Not Required

\hookrightarrow if we want then do Unification Surgery

\Downarrow
"STARSHMAN METROPLASIA"

* Unicornuate Uterus \hookrightarrow





Wt. → Multiparous (80 gm)
→ Nulliparous (50-70 gm)
↳ In pregnancy (term) = 1000 gm (1.1 kg)
1 kg

} dl + hypertrophy
↓
hyperplasia

volume of Non-pregnant = 10 mL } dl + hypertrophy >> hyperplasia
volume of Pregnant = 5 L

Q. Q. Weight of Uterus six week after Postpartum = 80 gm

* Body ⇒ Is Made up of Smooth Muscle

(Myometrium)

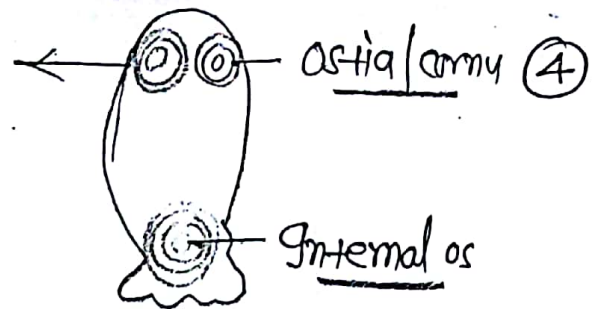
← Inside → ← Outside →

Endometrium serosa

Myometrium = 2-2.5 cm thick

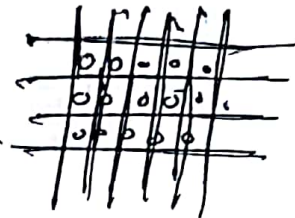
3 Layer ⇒ Inner - circular muscle
Middle - criss-cross
Outer - Longitudinal

Here Inner Muscle
works as sphincter



⇒ Middle Layer - criss-cross

work as Living Ligature **



⇒ Endometrium

↳ Gland + stroma

Simple tubular

↳ Single Layer columnar epithelium

cilia pres. only Near gland opening

↳ Superficial Layer = Functional ⇒ Shed off every Month
In Menses

Basal Layer = Not Shed off

↳ function = Regeneration

↳ so thickness of endometrium changes

Just after Menses (D5) = Thickness
0.5 mm

Proliferatory = 2-3 mm

Secretory = 5-6 mm

Implantation = 10-12 mm

vigorous curettage \Rightarrow

damage the basal layer



Result in Intrauterine Adhesion



Result in Asherman's Syndrome

Asherman's Syndrome

Highest Risk in to Manage the Post-partum hemorrhage

Post partum hemorrhage

Pt. is Amenorrhoea

infertility

Pt. is with Amenorrhoea infertility

\Rightarrow outside part of Myometrium = Serosa

Anteriorly Loose fold of Peritoneum \Rightarrow Uterovesical fold

Posteriorly Loose fold of Peritoneum \Rightarrow Rectouterine Pouch

* At what Level uterus opens -
into cervix? \rightarrow Internal os

Anatomical (Above)

Histological (Below)

0.5cm

\Downarrow constriction

\Downarrow Klu "Gsthmus"

* Gsthmus is part b/w Anatomical & Histological Internal os.

* Gsthmus — stretch — LUS

\rightarrow Lower uterine segment

In term LUS Gsthmus + cervix ; cervix comes above via
(not) (30%) cervical attachment (taking up
of cervix)

cervical effacement

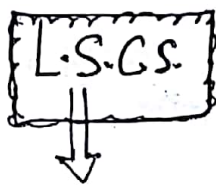
↳ Shortening + Softening

(5)

* At term LUS \Rightarrow 5cm

In Complete (Manual Labour) LUS = 10cm

* LUS shortly forming after 1st Primipara.



At Lower segment

How to identify

by

Loose fold

Peritoneal



Most common Incision \Rightarrow Low Transverse Incision



Kerr's Incision

Shape \Rightarrow Transverse or Pfannenstiel

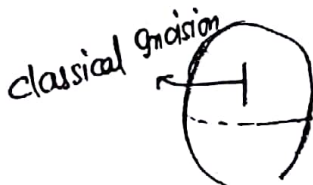
Klas "Kerr's Incision"



Kronig's Incision

Low vertical Incision

Klas "Kronig's Incision"



classical Incision

classical Incision

↳ Klas "classical Incision"

Risk of Rupture
Risk of Rupture

* Low transverse Incision \Rightarrow 0.2-0.9%

* Low vertical Incision \Rightarrow 1-7%

* Classical Incision \Rightarrow 4-9%
↳ Weakest Incision

* Classical Incision | scar is already given

↳ it is Absolute Indication for Repeat Cesarean
Section

but Not an indication for Repeat classical
Incision

* Indication of classical \Rightarrow ① ♀ 1st Caes.

② Dense Adhesion b/w bladder
& Uterus (May Injured bladder)

③ Repaired vagino-vertical fistula

④ Post Martem C.S.

N.B. Anterior Located Placenta previa (only if a Not trained enough
doctor)

↳ So; for Anterior Placenta previa \Rightarrow LSCS do *

* on other side of uterus 3 structures are attached

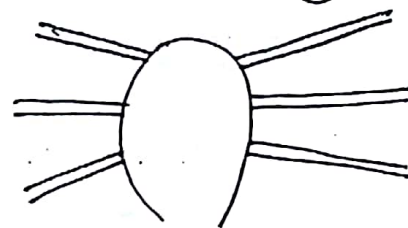
Mnemonics

↓
RTO

— Round Ligament

— Fallopian Tube

— ovarian Ligament



Adnexa ⇒ Fallopian tube + ovary

Antero-Posteriorly

⇒ Round Ligament → Fallopian tube

↓
ovarian Ligament

⇓
Utero ovarian Pedicle

Superio-Inferior

Fallopian tube

↓ below it

Round Ligament / ovarian Ligament @ same Level



They assist you in tubal Ligation Surgery

↳ M/c cause of failure of tubal

Ligation sx ⇒ Ligation of wrong structure

FUNDUS \Rightarrow Part of uterus lies above the attachment of Fallopian tube

Round Ligament Path \Rightarrow Upper Uterus \rightarrow deep Inguinal Ring

Pulling Uterus Anteriorly i.e. in Anteverted position.

\downarrow
Inguinal canal

\downarrow
Superficial Ring

\downarrow
Inverted on Labia Majora

* CANAL OF NUCK (Ent in Fetus only) :

Fold of Peritoneum in fetus that contains Round Ligament & extends into Inguinal canal

It carries Round Ligament.

* Round Ligament
Ovarian Ligament

developed from Gubernaculum



Not derived from Mullerian duct.

Proximal \Rightarrow Ovarian Ligament

Distal \Rightarrow Round Ligament

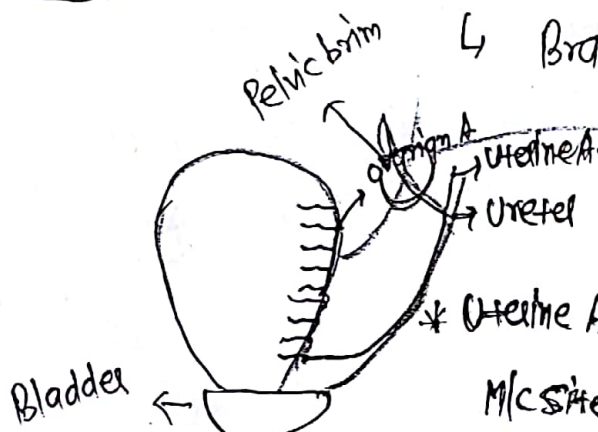
Blood supply \Rightarrow Uterine A.

\hookrightarrow Branch of Anterior division of Internal Iliac Artery.

2nd M/C site of ureteric injury

"Water under bridge" \Rightarrow Danger Area
is crossing over the ureter

\downarrow
M/C site for ureteric injuries



* Location of "Water Under bridge" Area \Rightarrow 2 cm Lateral to cervix¹³
OR
1.5 cm Lateral to fornix.

* Ureter is Posterior to Ovarian & Uterine Artery; ⁽⁷⁾
but it is Anterior to Internal iliac A.

\Rightarrow Branches of Uterine A \Rightarrow

U \Rightarrow Uterine A.

A \Rightarrow Arcuate branches \Rightarrow Supply outer 1/3rd of Myometrium

R \Rightarrow Radial branches \Rightarrow Supply inner 2/3rd of Myometrium

B \Rightarrow Basal \Rightarrow Supply Basal endometrium

S \Rightarrow Spiral \Rightarrow Supplies the Superficial / Functional endometrium

- Always do B/L Ligation & do @ the Level of Internal os.

* Uterine A gives a special branch \Rightarrow Sampsons A \Rightarrow for Round Ligament

- Nerve supply \Rightarrow T₁₀, T₁₁, T₁₂, L₁

L Pain during uterine contraction
travels via this Root

Pain Relay via \Rightarrow "Frankenhauser ganglion"

We give "Labour Analgesia" (Level of block for vaginal delivery

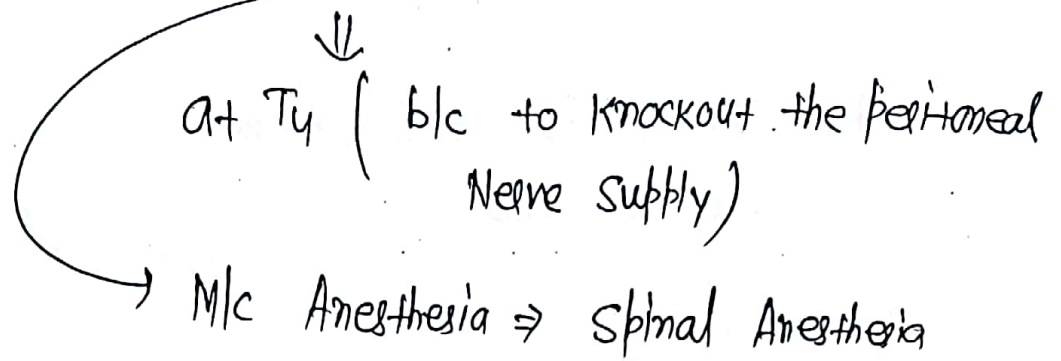
\hookrightarrow via Epidural Anaesthesia

give "Bupivacaine"

\hookrightarrow "0.125 - 0.25%" (Dose)

\hookrightarrow Sensory block; if given more
Motor block.

* Level of block for cesarean section



* Labour Analgesia may Prolong Labour (Active phase by 1hr)
↳ but doesn't ↑ Incidence of cesarean section,

* When we apply forceps (outlet/Low) - Pudendal N. Block

done only when complete
dilatation head @ +3.
to block it we

pierces Sacrospinous Ligament

↓
S₂, S₃, S₄

↓
Previously K/a

Direction of Needle (Posterior-Medial) "Saddle block"

* Lymphatic Drainage ⇒ Internal iliac + Ext. iliac L.N.

Fundus → Para-aortic L.N.

ostia → Superficial Inguinal L.N.

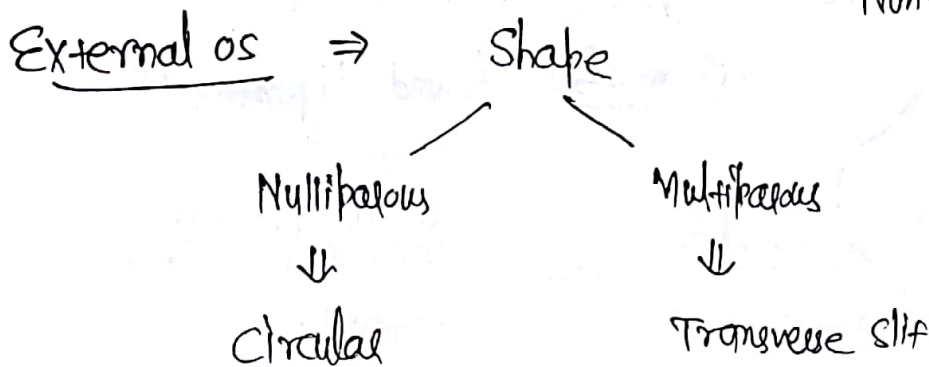
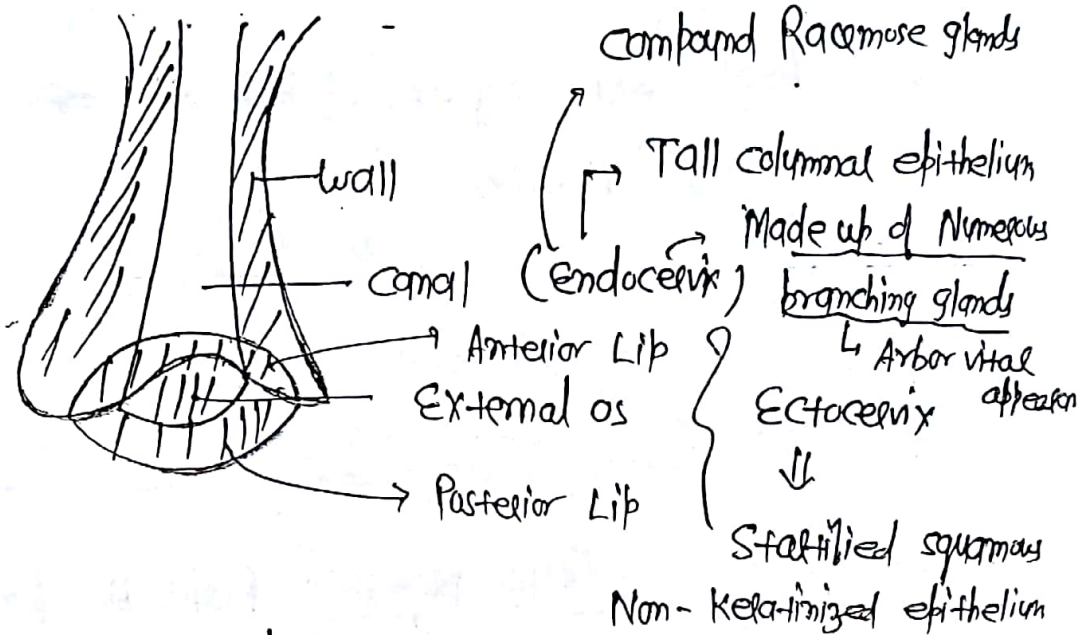
CERVIX \Rightarrow gt opens into vagina*

- Conical in shape

- 3cm Long

- cavity \Rightarrow spindle shape / fusiform

\downarrow
(a) external os** (8)



- wall of cervix \Rightarrow Made up of connective tissue (collagen)

\downarrow
10-15% Smooth Muscle

Result in effacement (there will be loss in collagen & its in hyaluronic acid / & its water content & its in dermis)

Softening & tearing up \Leftarrow

* Broad Ligament is a potential space; contains blood vessels etc.

* Angle b/w cervix & vagina \Rightarrow Anterersion
↓
90°.

* Angle b/w Long axis of body of uterus & cervix \Rightarrow Anteflexion
↓
120°
↳ @ Internal os

* In 80% women Anterverted & Antiflexed uterus ⊕
↓

Two Ligament Responsible for it

↳ Round Ligament

+

Uterosacral Ligament.



* If Fundus is More towards Bladder \Rightarrow Anteflexion

If Fundus is More towards Rectum \Rightarrow Retroflexion.

* In Plv examination \Rightarrow Which Lip you hitting 1st

Anterior Lip

↓

Anterersion

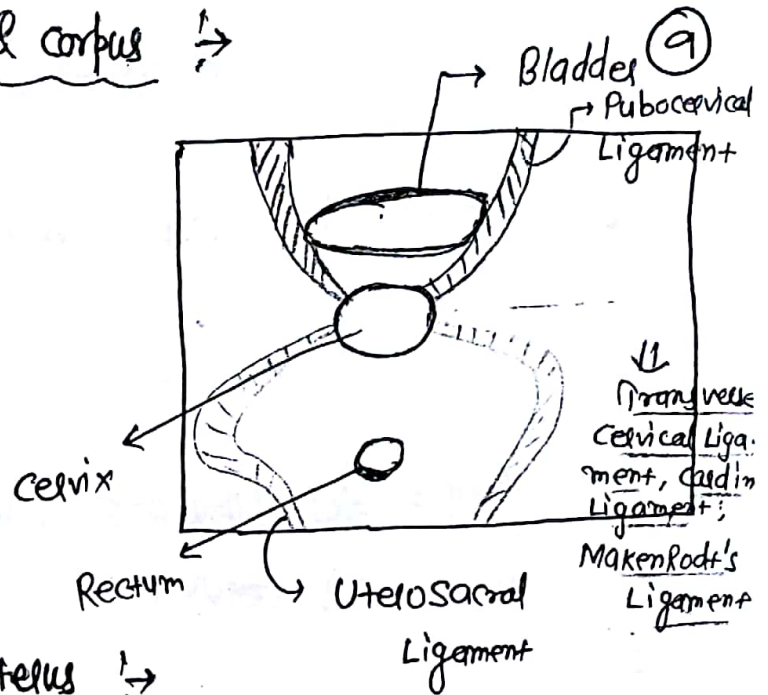
Posterior Lip

↓

Retroversion,

*^{**} Ratio of cervix & corpus →

At birth = 1:1
 Prepubertal = 2:1
 after puberty = 1:2
 Reproductive life = 1:3
 Post Menopausal = 1:1



* Main Support of Uterus →

Anterior ⇒ Pubocervical Ligament
 Posterior ⇒ Uterosacral Ligament
 Lateral ⇒ Transverse / cardinal / Mackenrodt's Ligament
 Inferior ⇒ Levator Ani

Hammock Ligament = Pubocervical Ligament
Uterosacral Ligament
Cardinal Ligament.

☹☹ All are Main Support except

↳ Round Ligament (support, but not main)

↳ Broad Ligament (Not a support)

↳ False Name

It is Nothing, but fold of Peritoneum

* Broad Ligament is a potential space; contains blood vessels etc.

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↓
90°.

* Angle b/w Long axis of body of uterus & cervix \Rightarrow Anteflexion
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Anterior Lip



Anterersion

Posterior Lip



Retroversion.

FALLOPIAN TUBE ^{KK}

(10)

- Unfused part of Mullerian duct*

- Length = 10 cm (10-12 cm)*

* Part of Fallopian tube (Medial - Lateral) \Rightarrow

• Intramural / Interstitial part (1-2 cm) = Narrowest part of Fallopian tube
 \hookrightarrow 0.7 mm diameter

• Isthmus ; (3 cm) = 1 mm diameter

• Ampulla ; (5 cm) \Rightarrow Widest part of Fallopian tube = 6 mm

• Infundibulum

\hookrightarrow Fimbrial end

\hookrightarrow Site of fertilization / M/c site of Ectopic



b/c here fertilization takes place & dth Mucosal fold
 Ent (a) Ampulla (Klas "plicae")

* M/c site of Ligation \Rightarrow Isthmus.

* Conceptus Remains in Fallopian tubes \Rightarrow 3 days

* Conceptus enter the uterine cavity on the 4th day.

\hookrightarrow Post-fertilisation

* Anatomical sphincter of Fallopian tube \Rightarrow Intramural part.

Physiological sphincter of FT \Rightarrow Isthmus

Q9

Main Reason for transport of conceptus

↳ Tubal Peristalsis *

Anything that ↓ Tubal Motility Leads to "Ectopic ⁺⁺⁺♀"

In Pelvic Inflammatory disease;

Tubal Surgery;

Progestelone only pills;

- epithelium of Fallopian tubes →

↳ Single Layer ciliated columnal epithelium.

3 cells - Secretory

ciliated

Peg cells → Resting cells of Fallopian tubes.

* Direction of ciliary Mucle is towards Uterus ⁺⁺

- Blood supply ⇒ dual blood supply *

Medial $\frac{2}{3}$ rd ⇒ Uterine A.

Lateral $\frac{1}{3}$ rd ⇒ Ovarian A.

↳ dilates 3 times in ♀
↳ site of Ligation for Management of ppy.

- Lymphatic drainage \Rightarrow Para-aortic Lymph Node*

(11)

Intramural + ostia

\hookrightarrow Superficial Inguinal Lymph Node*

extra edge

N. Supply \Rightarrow

T₁₁ T₁₂ L₁

\Downarrow

Pain Sensation from Unruptured ectopic
(tubal stretch)

* Ectopic is vascular accident
 \downarrow
die dlt \nearrow

VAGINA**

Embryological development \rightarrow

Upper 1/3rd of va from Mullerian duct (Mesoderm)

Lower 2/3rd of vagina \Rightarrow from Urogenital sinus (Endoderm)

\hookrightarrow from sinovaginal bulb

Hymen is Remnant of this \leftarrow

* Mucous Membrane of vagina \Rightarrow from Endoderm of urogenital sinus

* Muscle of vagina \Rightarrow from Mesoderm of Mullerian duct.

Vagina has four walls \Rightarrow Anterior
 \Downarrow
 7-10cm Long
 Posterior
 Lateral - 2 in No.

• Posterior wall is Longer than Anterior wall by 2cm.*

• A-P wall apposed together

\hookrightarrow after cut "H" shaped

* Cervix comes inside vagina & space b/w them k/as



Recess = Fornix

\Downarrow

Total 4 in No.

"Fornix"

\Downarrow

4 in No. (Ant, Post, 2 Lateral)

— Posterior fornix is deepest*

if More than 100ml collection in Pouch of Douglas \oplus

\hookrightarrow it is significant **

Pouch of Douglas / Cul-de-sac \Rightarrow

\Downarrow

Rectouterine fold Posterior
to Vagina

Culdocentesis

\Downarrow

if we get blood — which doesn't clot

\hookrightarrow Hemoperitoneum.

\rightarrow used in Ruptured ectopic.

if blood clot \Rightarrow then it comes from blood vessels.

Colpotomy \Rightarrow Opening of the Pouch of Douglas to drain
Pelvic Pus. (Abscess) (12)

Enterocele \Rightarrow Prolapse of bowel wall (Pouch of Douglas hernia)
 \hookrightarrow upper $1/3^{\text{rd}}$ of posterior wall

Q9. Which of the following is Cystocele?

a) upper $2/3^{\text{rd}}$ of wall;

b) Lower $2/3^{\text{rd}}$ of wall;

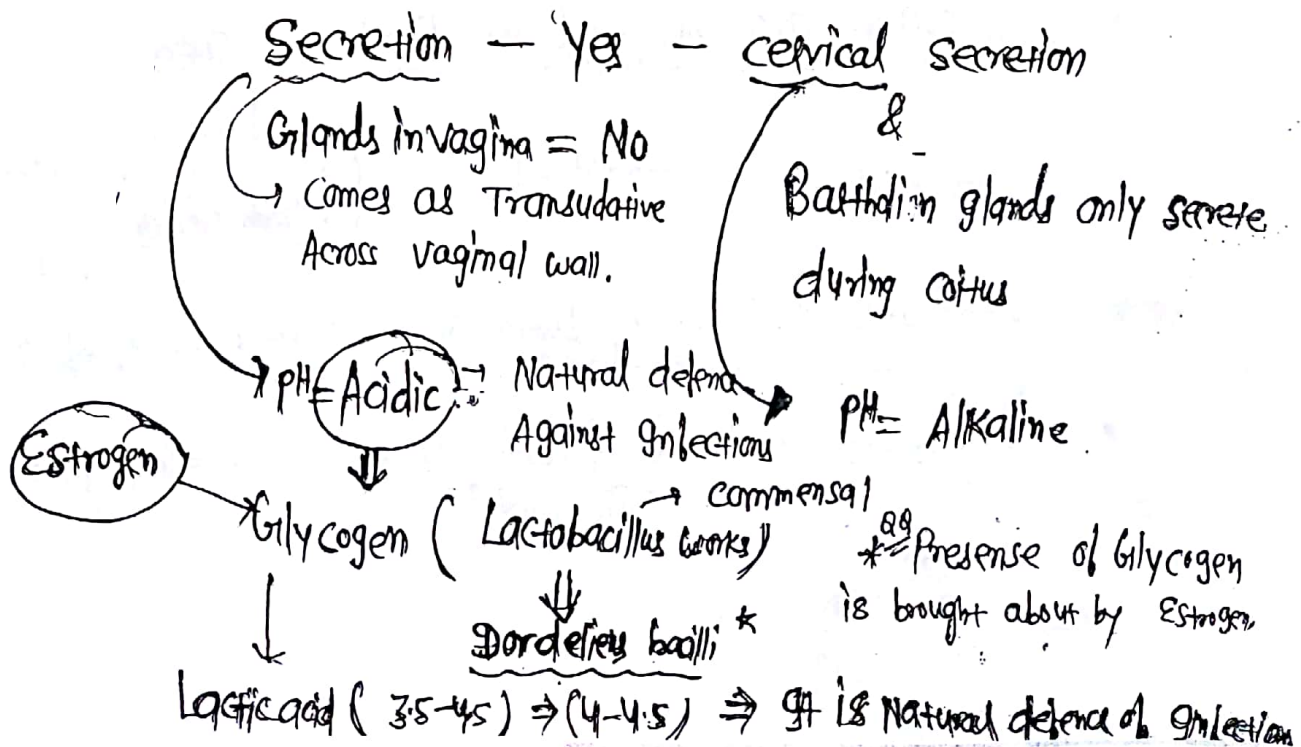
c) upper $1/3^{\text{rd}}$ of wall; * Rectocele \Rightarrow Prolapse into Middle
 $1/3^{\text{rd}}$ of posterior vaginal wall

d) Lower $1/3^{\text{rd}}$ of wall \Rightarrow Urethrocele

\hookrightarrow Protrusion into Lower one-third
of Anterior vaginal wall.

* \approx the H₃-line passing through Pelvis, vagina forms \Rightarrow 45°

* Epithelium of vagina \Rightarrow Stratified Squamous Non-Keratinized



Q. Q.

Deficiency of 5 α Reductase \rightarrow

Male Pseudohermaphrodite (Genotype - Male
Phenotype - Female)

* by looking external genitalia we differentiate Male & female by 12 weeks.

* Female Bartholin's gland Homologous Male Bulbourethral gland (Cooper's gland)

* Glands of Skene Homologous Prostate.
(Para-urethral)

* Lymphatic drainage of clitoris \Rightarrow Superficial Inguinal Lymph Node

* Lymphatic drainage of Glans clitoris \Rightarrow Deep Inguinal Lymph Nodes
(Lymph Nodes of Cloquet)

* Lymphatic drainage of Labia Minora glands \Rightarrow Deep Inguinal L.N.

* Lymphatic drainage of Labia Majora glands \Rightarrow Superficial Inguinal L.N.

Vaginal partBlood supplyLymphatic drainage

(13)

Upper 1/3rd

Descending Uterine Artery

External + Internal iliac

Middle 1/3rd

Inferior vesicle Artery

Internal iliac

Lower 1/3rd

Middle Rectal Artery

Superficial Inguinal

EXTERNAL GENITALIA

Genital tubercle → @ 6 weeks

Female

Clitoris

" Fold

L. Minora

" Swelling

L. Majora

Absence of
DHT Require for
Female like organs.Male (Homologous structure in Male)

Penis

Penile urethra

Scrotum

To convert in Male form

Testosterone

5 α -Reductase↓
Di-hydrotestosterone
↳ Tubercle / Fold / Swelling.

99

Deficiency of 5 α Reductase \rightarrow

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* Lymphatic drainage of Labia Minora glands \Rightarrow Deep Inguinal LN

* Lymphatic drainage of Labia Majora glands \Rightarrow Superficial Inguinal LN.

VESTIBULE

⇒ Anteriorly ⇒ Clitoris

(14)

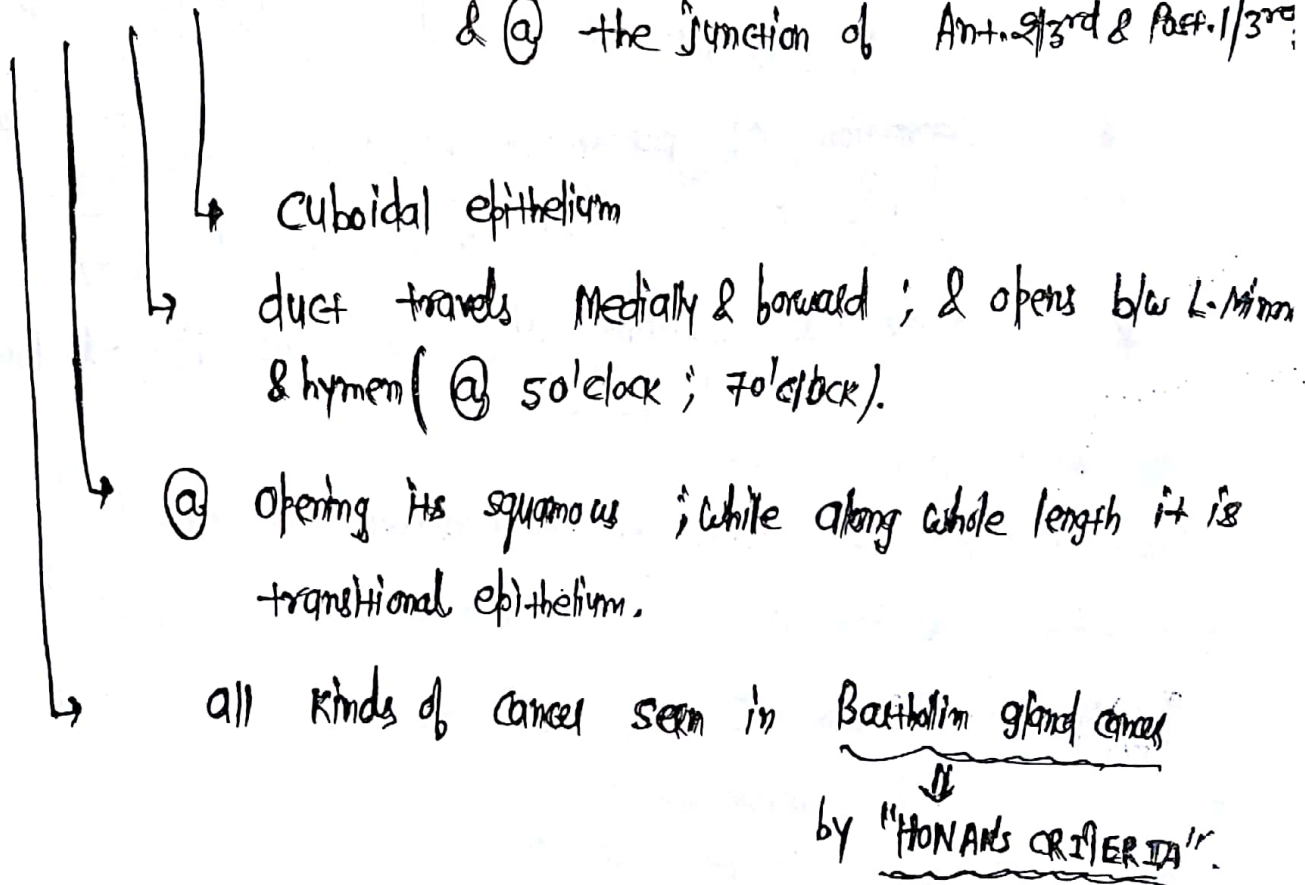
Posteriorly ⇒ Fourchette

Lateral boundary ⇒ Harts line

⇒ Structures opening in vestibule ⇒ Urethral opening;
Vaginal opening;
2 Bartholin's gland opening;

* Bartholin's gland cyst ⇒ Pea Size;

Bartholin's gland ⇒ Located b/w L. Minora & L. Majora groove
& @ the junction of Ant. 2/3rd & Post. 1/3rd



i) Asymptomatic Bartholin's cyst $\oplus \Rightarrow$ No Rx *

ii) Symptomatic / Recurrent cyst $\oplus \Rightarrow$ Marsupialization

\Downarrow
Exteriorization of the duct to prevent Recurrence.

iii) ≥ 40 yr & have Bartholin's cyst $\oplus \Rightarrow$ Excision
 \Downarrow
↑ Cervical Risk *

Bartholin's Abscess \Rightarrow i) E. coli > Gonorrhoea;
ii) Initial Tx = I & O

\downarrow
Marsupialization on Later date

* Secretion of Bartholin's gland \Rightarrow Alkaline; \rightarrow Released during coitus
b/c Acidic environment is spermicidal.

* they are content of Superficial Perineal Pouch;

RELEVANT EMBRYOLOGY

29

(15)

* Glands 21 \Rightarrow develop by Genital Ridge - 5 weeks ⁹⁹

Ovary (8 weeks) *

Testis

• Absence of Y chromosome

• Y chromosome

In XO genotype \Rightarrow ovaries appear Normally

• SRY functional part of Y chromosome

SRY \Rightarrow For testis

WNT4 \Rightarrow For ovary

ABN

functional

@ 6-7 weeks

Distal segment

b/c "XX" genotype of short arm
Require for function. of Y-chromosome

if Nothing Mention;
differentiation b/w σ & ϕ

@ 12 weeks OF P.O.G.

Streak Ovaries

\Rightarrow Seen in Turner Sx. *

* Glands can be differentiated into σ & ϕ by 7 weeks

* Internal genitalia differentiates @ 10 weeks;

* External genitalia differentiates @ 12 weeks;

- Ovary prt. in "Ovarian Fossa"

\Rightarrow prt. in Lateral Pelvic wall

* Posterior to ovarian fossa \Rightarrow Ureter; Internal iliac vessels;

Anterior to ovarian fossa \Rightarrow Obliterated Umbilical Ar.; Mesovarium

Lateral to ovarian fossa \Rightarrow Obturator Nerve & vessels

Pain from ovary Related to Medial aspect of thigh b/c of Cutaneous br. of Obturator Nerve.

Medial \Rightarrow Ovarian Ligament

Superior \Rightarrow External iliac vessels.

Inferior \Rightarrow Levator Ani

Ovaries \Rightarrow 3 Supports

\rightarrow Ovarian Ligament Ovarian ligament

\rightarrow Infundibulo pelvic Ligament Infundibulum pelvis ligament
(Suspensory Ligament)

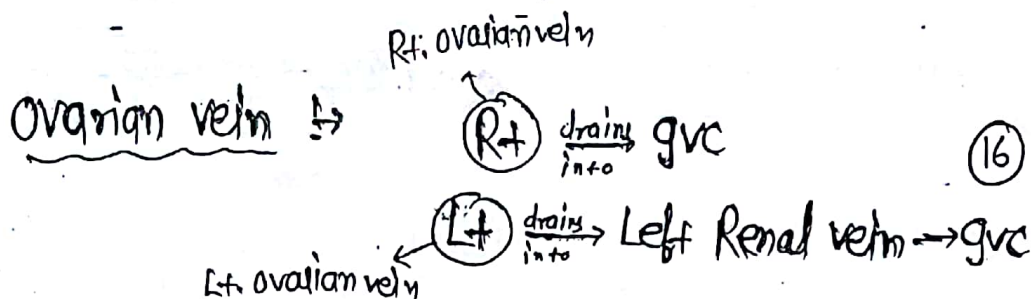
- Ovarian vessels (gt carries ovarian A. & veins)
- to Lateral Pelvic wall (Attaches ovary to Lateral Pelvic wall)
- Cut - for oophorectomy

\rightarrow Mesovarium - False Ligament False Ligament
Mesovarim

\rightarrow Fold of Peritoneum
to posterior leaf of Broad Ligament.

OVARY \Rightarrow In Reproductive age Average 7-8 cc upto 20cc Normal.
in Postmenopausal age average 2-3 cc upto 10cc Normal.

Blood supply \Rightarrow * Ovarian Artery \Rightarrow br of Abdominal Artery
@ L2



Varicocele \Rightarrow M/c on left side; b/c ~~it makes 90°~~ it makes 90°

\hookrightarrow M/c Reversible cause of Male Infertility

(Lymphatic Drainage \Rightarrow Para-aortic group of \wedge Lymph Node)

Epithelium \Rightarrow Germinal epithelium

(Single layer cuboidal)

\hookrightarrow Germ cells are formed here

1° Germ cells (Primordial Germ cell)

\hookrightarrow Epiblast (Ectoderm) \Rightarrow older days from Yolk sacs

* Ovary has

- Cortex — Follicles
- Medulla — vascular

* Menopause \Rightarrow Follicles goes into Programme cell death cause it

\hookrightarrow Epiblast \rightarrow Maxim follicles @ Intrauterine life 5th month (20 week)

\downarrow Yolk Sac (@ 3 weeks)

\downarrow Genital Ridge (@ 6 weeks)

\downarrow Oogonia (@ 9 weeks)

\downarrow 1° oocytes (@ 12 weeks)

\downarrow Follicle formation begins (@ 14 weeks)

\downarrow Follicle formation complete (@ 24 weeks)

on Birth = 2 million (1-2)

on Puberty = 400,000

ovulate = 400 ovulate

1000 atresia / Month

Follicle / oocyte

Folliculogenesis

Folliculogenesis

Oogenesis

Oogenesis



What is happening
in surrounding cells



What is happening
in germ cells

1° Follicle - Flattened
granulosa cell



1° Follicle - cuboidal
granulosa cell.



2° Follicle - Theca cells
⊕

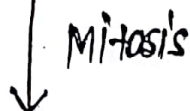


Antral Follicle - Cavity ⊕

* all follicles have 1° oocyte @ centre; but ovum have
2° oocyte.

* oogenesis begins IUL ⇒ @ 9 weeks

2n Oogonia

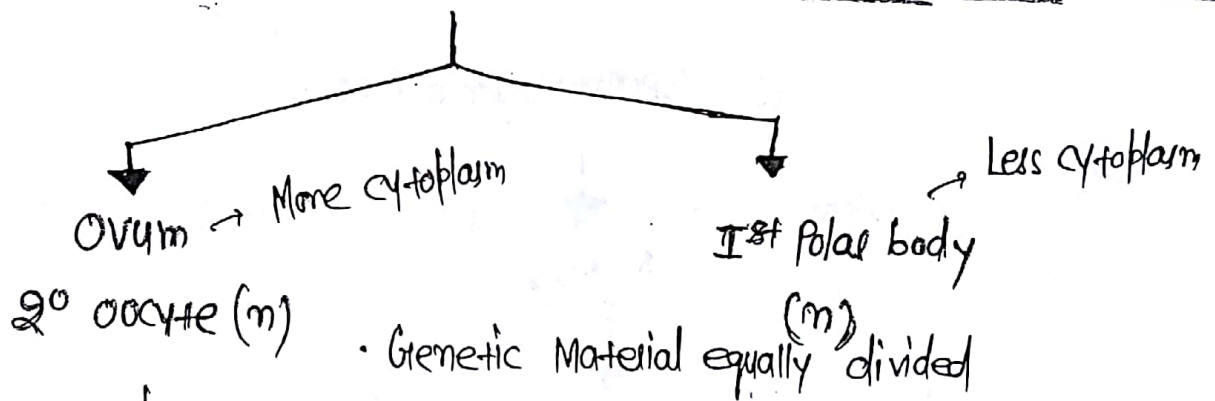


1° oocytes - diploid



Meiosis I

(17)

Meiosis - IArrested In \Rightarrow Prophase↓
diplotene↓
* Dicryotene (Stage b/w Prophase & Metaphase)
* Arrested stageWhen completed?? - just before ovulation
@ ovulation (3-4 hours before ovulation)↓
Enters into Meiosis - II

Arrested - II - Metaphase *

Completed - Ab-tel fertilization

Female ProNucleus *

2nd Polar Body

Fig. Fertilized ovum ** Life span of ovum = 24 hr.

* Size of Mature ovum = 120 μ m in diameter

Size of Mature follicle = 18-20mm in diameter

Size @ which follicle Rupture = 18-20mm *

SPERMATOGENESIS

* Begins — Puberty in seminiferous tubules.

* duration — 72 day

① Spermatogonia (2n)

Mitosis

② 1° Spermatocyte (2n)

Meiosis-I Each (Dictyotene absent) *

2° Spermatocyte

Meiosis-II

Spermatids

Spermatids

2° Spermatocyte

Meiosis-II

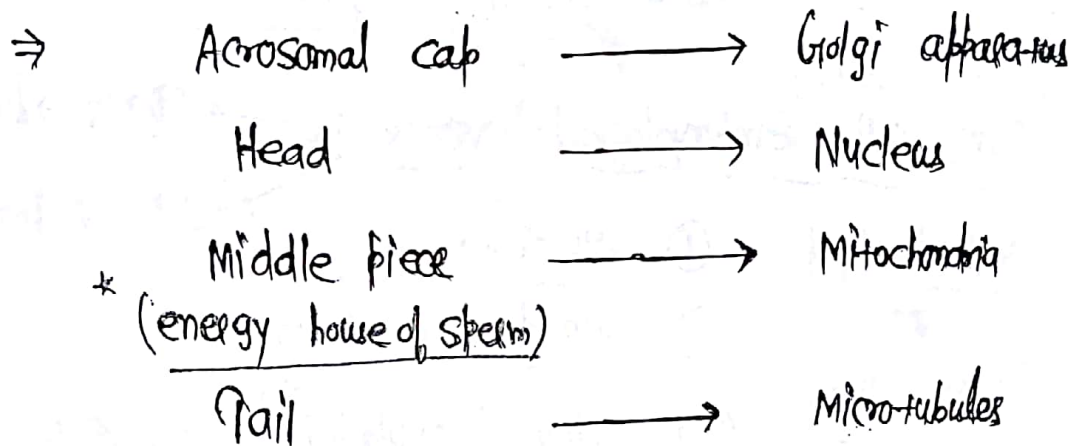
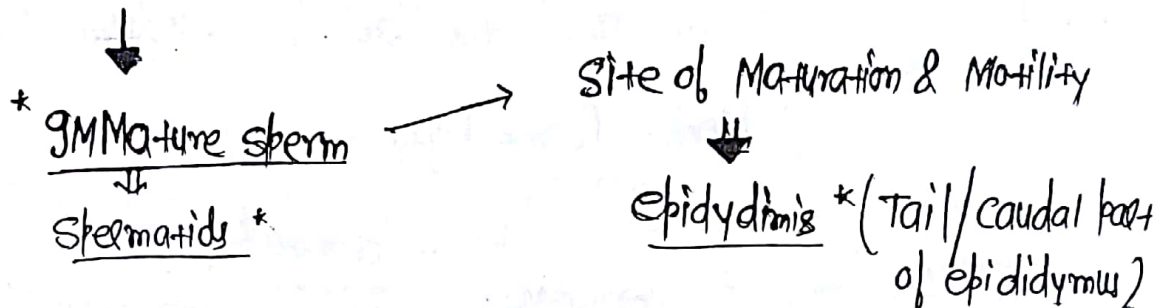
Spermatids

Spermatids

- * One 10^6 spermatocytes gives \Rightarrow 4 sperm = 4 spermatids.
- * one spermatogonia gives \Rightarrow $\frac{16 \times 10^6 \text{ spermatocytes}}{4} = 64 \text{ sperm}$
- * Fertilizable Life span \Rightarrow 3 days; (18)
- * Mature sperm \Rightarrow 5.5 μ m length (Smaller than ovum)

* No. of Sperms produced in one day = 100 million *
(Average sperm count)

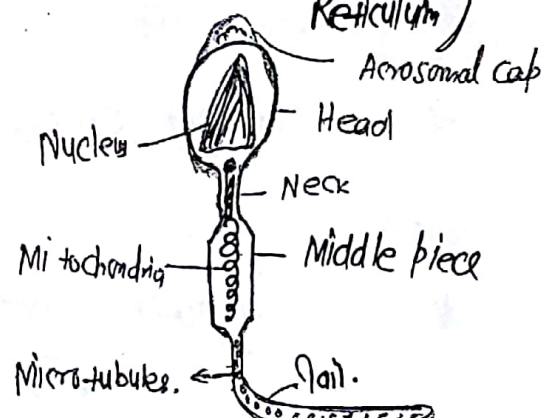
* Spermiogenesis \Rightarrow 12-14 days



Sperms don't have ^{aa} Endoplasmic Reticulum (Rough endoplasmic Reticulum)

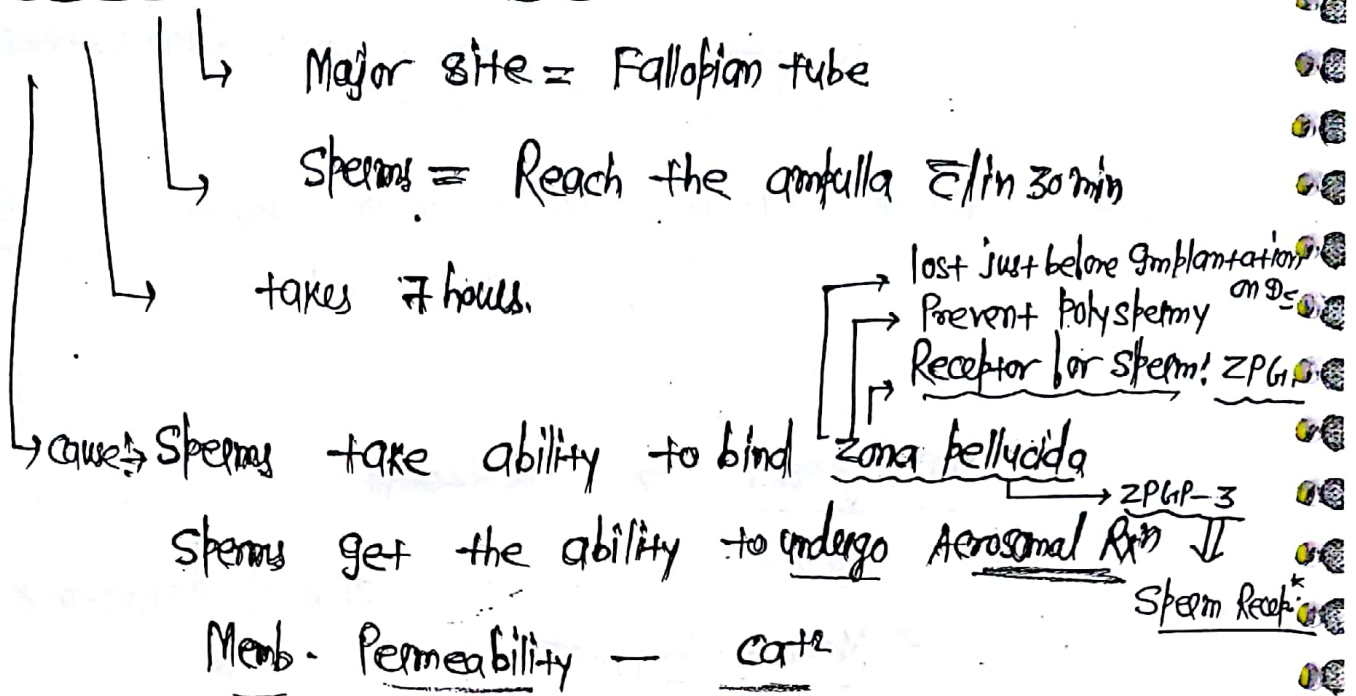
* Ion Responsible for Motility of sperm \Rightarrow Calcium ^{aa}

* Gene for Motility \Rightarrow CATSPER



FERTILIZATION

Capacitation \rightarrow begins in cervix*



* Acrosomal Rxn \Rightarrow Main Enzyme! Hyaluronidase^{aa}

* For all Embryological events \rightarrow Days \Rightarrow from Fertilization

\rightarrow Weeks \Rightarrow from LMP (1st day)

- 3 Rules \Rightarrow
- ① 28 day;
 - ② 14th day - ovulation
 - ③ days of ovulation = days of Fertilization

* 1st cleavage occurs \Rightarrow 20-30 hour after fertilization

* Conceptus enters in uterus \Rightarrow Morula stage @ 4 days

\downarrow

16 cell stage \rightarrow 8 cell stage

* Implantation occurs \Rightarrow in Blatocyst form

\rightarrow on D6 begins (D6-D7) \rightarrow on D5 it form

* Implantation occurs in 3 Phases ! →

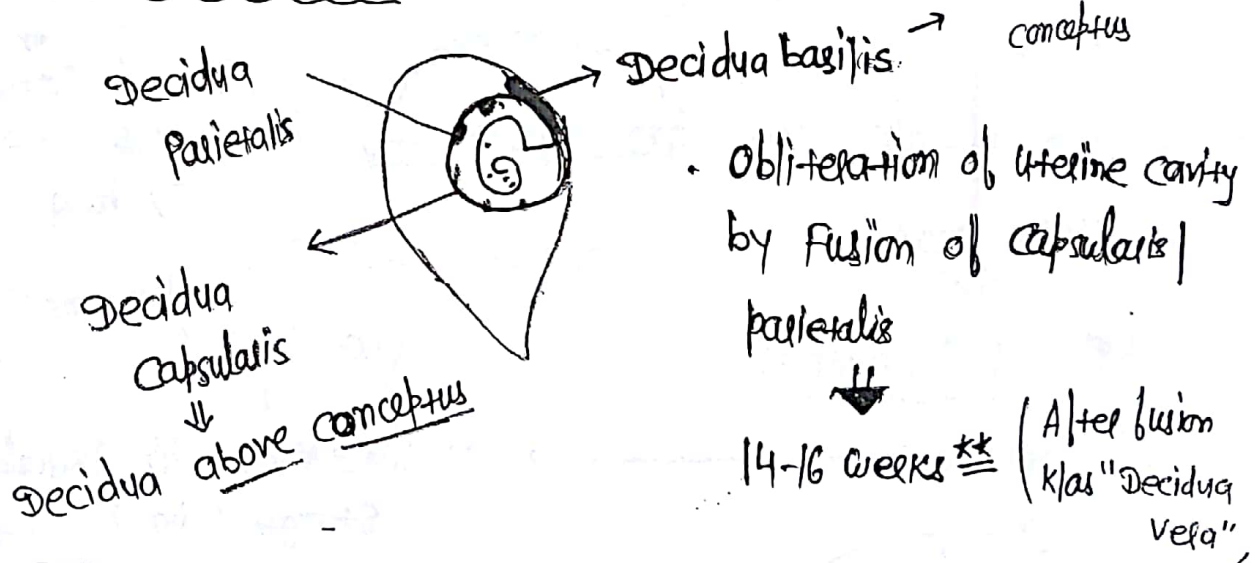
(19)

1. Apposition → Selecting
2. Adhesion → Integrating =
3. Invasion → Matrix Metalloproteinase.

* Implantation completed @ D10.

* M/c site = upper posterior wall (Eccentric)
↳ one half bigger than other side

* Endometrium of ♀ → klas "Decidua" *



SUPERFETATION

- Fertilization of 2 ova by 2 different sperms by 2 different acts of coitus

Different

↑
Menstrual
cycle

Not documented
in Human

- Theoretically twinning can happen upto 16 weeks.

SUPERFECUNDATION *

↑

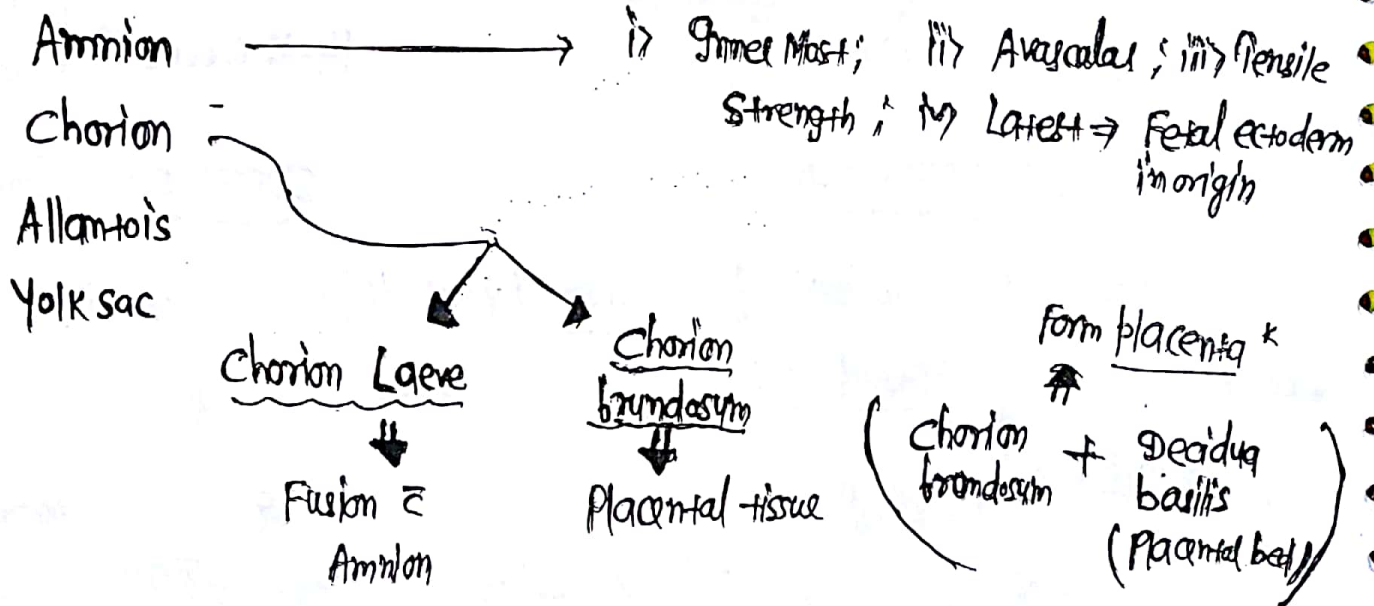
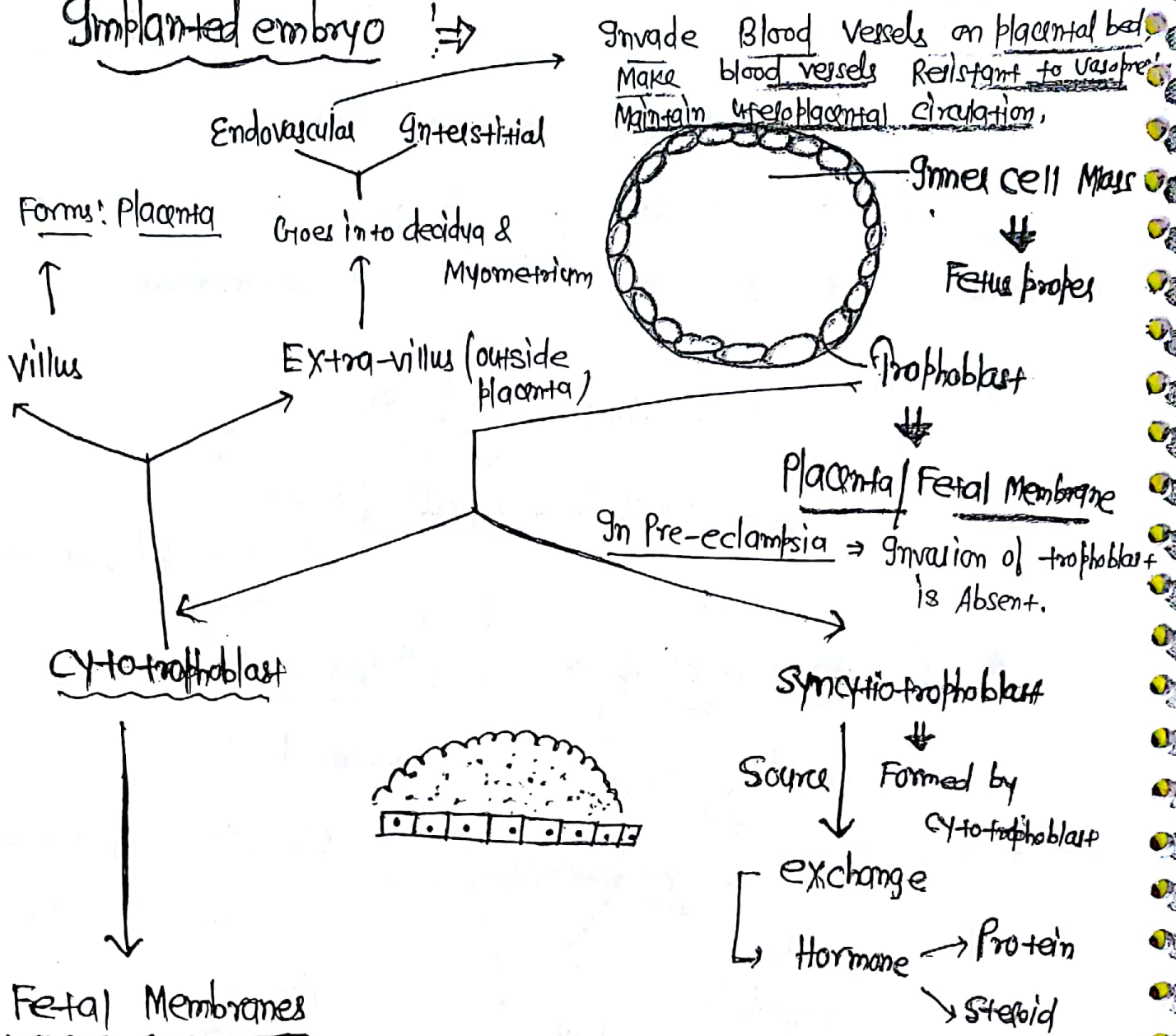
2

Same

Menstrual cycles

* Embryonic phase lasts upto 8 weeks post fertilization & 10 weeks from L.M.P.

Implanted embryo ⇒



Allantois \Rightarrow Diverticulum from hindgut grows into connective
Stalk. (20)

\rightarrow Umbilical vessels (It gives rise Umbilical vessels).

Yolk sac \Rightarrow 1st site of hematopoiesis

\rightarrow 3rd - 6 weeks (Portland, Golfer 1; Golfer 2)

\rightarrow 6 weeks - Linear \Rightarrow HbF $\Rightarrow \alpha_2\gamma_2$

\rightarrow 20 weeks - Long bones

• Bigger in size & shorter in life span.

• Has 2,3 DPG & carbonic Anhydrase; Hb-O₂ curve shift to Left.

* Fetal Hb is Resistant to both alkali & Acid denaturation

ALKALI DENATURATION TEST

APTT (test)

\downarrow

1% NaOH used

ACID DENATURATION TESTS

• KB (Kleihauer Betke)

• Citric acid & Ph buffer used.

Bed side test

Qualitative test

\downarrow

differentiate Maternal blood & Fetal blood from each other

Mom's \Rightarrow \ominus ve (colour change)

Baby's \Rightarrow \oplus ve (colour Resistant)

• Laboratory test

• Quantitative test

\downarrow
Fetal RBC from Maternal RBC \rightarrow count

• Singer's test (other kind of alkali test)

\downarrow
Used in Rh \ominus ve \rightarrow to calculate dose of Anti-D.

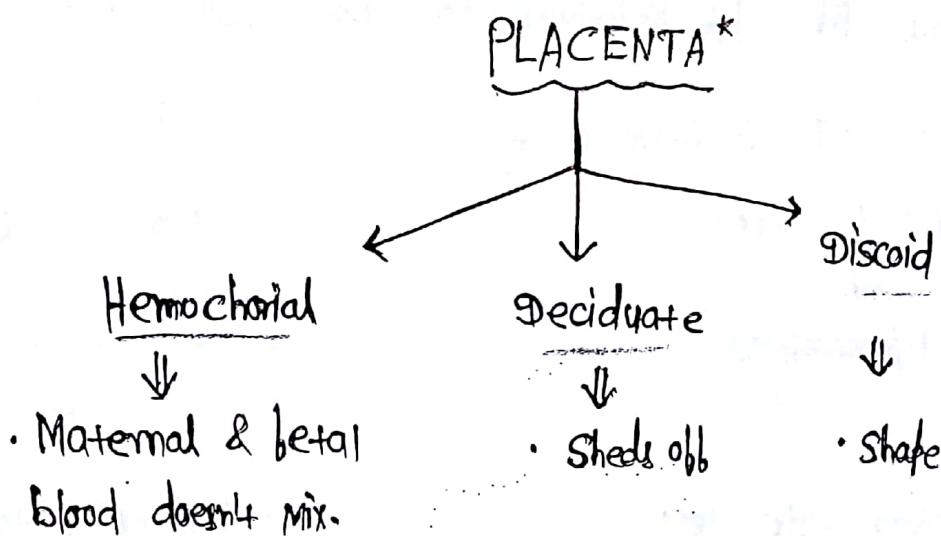
In Antepartum hemorrhage we can do "APT test". *

* Fetal RBC \Rightarrow Larger in size
Shorter Life \Rightarrow 90 days *

- Hb of baby @ birth \Rightarrow 18 gm%.

75-80% of HbF & Rest Adult Hemoglobin.

- @ 6 Month $<$ 1% HbF pres. out of total.



Wt of term placenta \approx 500 gm

Volume of term placenta \approx 500 mL (volume)

Diameter \approx 20 cm

Thickness \approx 2.5 cm

At term; Placenta: Fetal wt \approx 1:6

Maternal side

Fetal side

↓
Facing decidua

↓ (2)
Facing the fetus

↓
Lobes (divided into 15-20 lobes)

Identify ⇒ Smooth / shiny
(b/c of Membrane)

↓ each lobe divided into 3-5 lobules
Lobules (Functional unit)
↳ like "cotyledon"

Umbilical cord attached

↓
@ centre of Placental disc

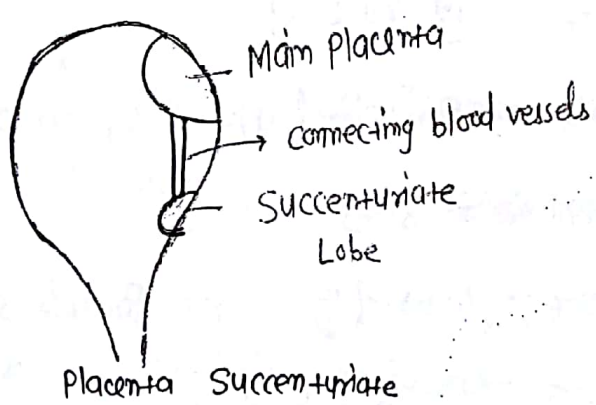
* Placenta bilobata ⇒ Placenta is separated into lobes;

• Division is incomplete & the vessels of fetal origin extends from one lobe to the other before uniting to form umbilical cord.

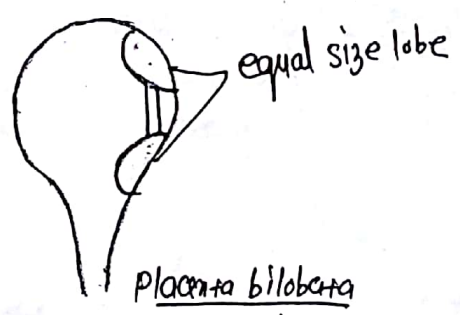
• if cord attached to the periphery ⇒ Battledore Placenta

* Placenta Succenturiate ⇒
↳ having Accessory Lobe; which is connected to the main part of placenta by blood vessels.

↓
It can cause Post partum Hemorrhage



• Velamentous Placenta ⇒ if fetal vessels travel outside the cord for some distance before reaching placenta (∴ More chance to injury).



• Vasa previa ⇒ vessels are travelling over the internal os.
type of velamentous placenta;
Rare type of APH (Fetal blood loss).


CS do ← Obstetric emergency ← Result in severe Fetal stress

* Intervillous space \Rightarrow Maternal blood*

Inside villi \Rightarrow Fetal blood*

1° villous \Rightarrow  \rightarrow Solid — D₃

2° villous \Rightarrow  \rightarrow Mesodermal core — D₁₆

3° villous \Rightarrow  \rightarrow Blood vessel — (D₂₁)

* Fetal blood flow through placenta \Rightarrow 400 ml/min.

Fetal circulation Established @ D₂₁

Uteroplacental circulation = D₁₂

@ term = 450-650 ml/min.

* Intervillous space \Rightarrow 140 ml blood

\downarrow Po₂ in Intervillous space \rightarrow 120 spiral arterioles @ Inside.

\downarrow
- 35-40 mm of Hg.

O₂ Saturation = 65-75%

Low pressure = 10 mm of Hg

\rightarrow Invade spiral A

Klas "endovascular"

* Cytotrophoblast

Extravillous trophoblast

Syncytiotrophoblast

Cytotrophoblast

Maternal blood

\downarrow
Permanent vasodilation

\downarrow
Good uteroplacental circulation

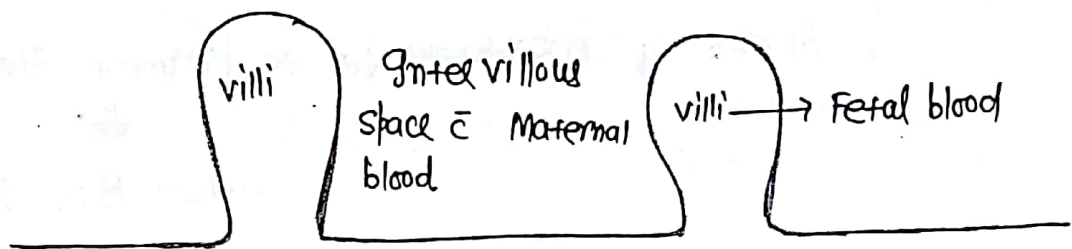
\downarrow
Vascular Remodelling

* Vascular Remodelling is controlled by decidual Natural Killer cells. (22)

* Completed 2 phases — 12 weeks
— 16 weeks

* Absent Vascular Remodelling \Rightarrow Pre-eclampsia
JUGR

* (ABN) Vascular Remodelling \Rightarrow Adherent Placenta*



PLACENTAL FUNCTIONING*

* Placental formation begins @ 6 weeks.

Anatomically — Placenta completely formed by 16 weeks.

Physiologically — Maturation continues

\uparrow POG \Rightarrow • Cytotrophoblast \downarrow term
• Syncytiotrophoblast Thickness \downarrow (thin)
• \downarrow Stroma
• \uparrow Fetal blood vessels.
also keep moving towards periphery of villus
• Hofbauer cells \oplus
 \hookrightarrow Fetal Macrophages

No cytotrophoblast @ term

Function of Placenta →

Nutritional

Excretory

Respiratory

Endocrinal (Most imp)

Progesterone

Estrogen

HPL

HCG

Human Placental Lactogen

Progesterone ⇒

Maintenance of ♀

↳ Smooth Muscle contracts

↳ Amount of progesterone ↓ ⇒ Recurrent Abortion

↓
Luteal Phase Defect (L.P.D.)

also do decidualization (Hypersecretory change)



On HPE ⇒ "Arias Stella Reaction"

↳ No Progesterone



No Arias Stella Reaction.

* Source of Progesterone in early ♀ ⇒ Corpus Luteum

Rescue the corpus luteum from Luteolysis

↓
Pregnancy

HCG

Maintain corpus luteum survive

Pregnancy

Natural late Luteolysis *

* Placenta will take over the function of corpus luteum. 23

↓
8 week (8-10 week)*

→ Make Progesterone by precursor \Rightarrow Maternal LDL cholesterol

* corpus luteum of pregnancy will Regress

→ ~ 12 weeks

* Ovarian cyst of 1st Trimester \Rightarrow May be enlarged corpus luteum
→ Observation & Resolve after 12 weeks.

Estrogen \Rightarrow Specific to ♀ = E_3 Estriol*

E_2 = also formed; but Not specific

→ Growth of Uterus

→ Mask of ♀ = Melasma

↓
Hypopigmentation

(Stimulates Melanocytes)

→ Retain salt/water

→ obstetric cholestasis

→ Thyroxine binding globulin ↑

* Placenta can't synthesise estrogen on its own

↳ b/c 17 α hydroxylase Lacks
↳ dependent on the fetus for synthesis of estrogen.

↳ Fetal DHEAS (from Adrenal glands)



Placenta



Sulphatase
Aromatase

→ Estrogen (E_3)



Tells about fetal well
being

HPL (Human Placental Lactogen) | HCS (Human Chorionic Somatomammotropin)

⇒ It tells about i) Placental functioning



As P.O.G. rises ⇒ Maternal ⇒ ↑ HPL

Peak = 36 wks.

Q9 Which hormone is produced by placenta in max^m Amount
at term = HPL (1gm/day).

ii) endocrine function ⇒ Main function

↳ Insulin Resistance in ♀
(Placenta secretes: Growth hormone)

Q. Q. Which hormone is responsible for fetal growth

(24)



Insulin like growth factor/ IGF

* Promotes Maternal Lipolysis - Levels of free fatty acids are low which mother utilizes as a source of energy; sparing glucose for fetus.

HCG Hormone ⇒

Glycoprotein hormone

(Human Chorionic Gonadotropin)
Identical

α/β

β specific

α Subunit ⇒ LH / FSH / TSH
↳ Non-specific

Syncytiotrophoblast

as early as 8 days post fertilization (ovulation)
Secretes "HCG" (8-9)

aa
→ This hormone has highest carbohydrate content of any human hormone

↳ We want to take hCG before 8 days

↳ do serum hCG ⇒ Quantitative
Very sensitive

↳ detect 1 IU/L

Urine hCG ⇒ Qualitative

↳ 20 IU/L ⇒ +ve after missed period.

based on "Sandwich ELISA"

* Serum Sensitivity ⇒ F.I.A. > R.I.A. > ELISA = RRA > IRMA

Fluorescent
Immunoassay

Radioimmuno
assay

Immuno Radio-
Metric Assay.

Radio Receptor
Assay

⇒ hcg Value rises as P.O.G. rises

Max^m = @ 10 weeks*

After 10 weeks = ↓

Min^m = @ 16 weeks

After 16 weeks = Plateau

→ normal rise
Doubling time of β -hcg

↓
48 hrs.

→ it means rise in 55% - 66%
Min^m after 48 hrs.

→ it does N't mean 100%
or double

v.v.g.
Q9

1st D_1 / D_3 - rise by 55% ?

(N) Intrauterine pregnancy

2nd D_1 / D_3 - rise by < 55% ?

Ectopic @

3rd D_1 / D_3 - ↓

Non-viable @ / Abortion

* Critical value of hcg for TVS = 2000 IU.

TAS = 6500 IU.

If hcg is more than or equal to these values & we don't see
sac in the uterus → Likely to be ectopic @.

(25)

* Condⁿ where hcg is less than Expected

- Multifetal ♀
- GTD (Gestational Trophoblastic Disease)
- Down's Syndrome
- Hyperemesis gravidarum
- Underexpected Gestational Age.

* Condⁿ where hcg is less than Expected

- Non-viable
- Ectopic ♀
- Overexpected gestational Age
- Trisomy 18.

- Functions of hcg ⇒
- Maintenance of ♀
 - ↓ uterine contraction
 - Growth & development of umbilical cord
 - 1st stimulus for Release of testosterone from Male foetus - hcg
 - Immunosuppressant

Q. Why the conceptus Not Rejected?

- i) Villous trophoblast Lacks HLA (MHC);
- ii) EVT (Extra villous trophoblast) - that have HLA/GI → only in Human seen Immunosuppressive

Placenta decidua \rightarrow NK cells have deficient cytotoxicity.

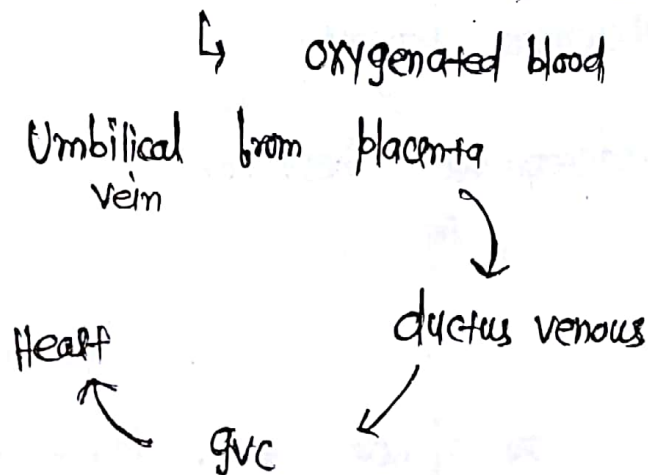
\rightarrow M/c Insertion \Rightarrow center.

UMBILICAL CORD (Attached to fetal side of Placenta).

- Average Length = 55cm (30-70cm)

- 3 vessels \Rightarrow 2 Umbilical A. (deoxygenated blood)
1 Umbilical vein (Left) "Right U-vein disappears"

* Short cord < 30cm
* Long cord > 70cm



* Max^m O₂ saturation pr^t in \Rightarrow Umbilical vein
 \downarrow
80%.

* M/c Vascular Anomaly \Rightarrow SUA (2 vessel cord)
(Single Umbilical A.)
 \downarrow then check
GCA (Gross Congenital Anomaly)
 \rightarrow CVS;
 \downarrow
M/c gross Anomaly

(Renal) \Rightarrow M/c Anomaly
 \times
Not gross \leftarrow SUA

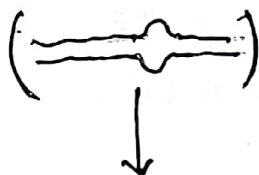
* if we see SUA + GCA

↓
↑ Risk of Aneuploidy*
(Trisomy 18)**

(26)

* Isolated SUA ⇒ doesn't ↑ Risk of Aneuploidy*

* Incidence of SUA is higher in ⇒ Twin ♀ / Multifetal ♀



↑ Risk of true knot of Umbilical cord.

False knot ⇒ Protrusion of Wharton's Jelly containing loop of umbilical vessels.
↓
No clinical significance

True knot ⇒ ↑ the Risk of still birth;

↓
cause ⇒ Fetal Movement.
↓
d/t Active fetal Movements
↓
↑ in Twin.

1st Umbilical Artery



Umbilical vein



Ductus Venosus



Ductus Arteriosus



Foramen ovale

DOWN'S SYNDROME *

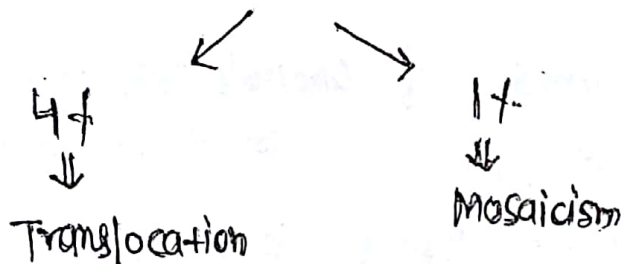
• Trisomy 21.

• Causes \Rightarrow Most common cause * \Rightarrow Non-dysjunction (95%) $\xrightarrow{\text{during Meiosis-I}}$
 \Downarrow
It is by chance; so;
Non-hereditary in Nature.

• Recurrence = 1%.

• H/o 1st baby down's \rightarrow Antenatal testing done in Next @.

• 5% \rightarrow Inheritable



18+ baby \rightarrow Down \rightarrow (Translocation 21, 21) $\xrightarrow{\text{Robertsonian Translocation seen in Down Sx.}}$

\downarrow
Risk = 100%.

\rightarrow Abortion. (in 2nd baby)

* All @ Women

\rightarrow offered down's screening. (opt-out screening).

SCREENING →1st Trimester

NT $\geq 3\text{mm} \Rightarrow \oplus$ ve \leftarrow USG

(Nuchal ~~translucency~~)
 ↳ Fluid collection behind Neck;
 • 11-13⁺6 weeks

Dual test \Rightarrow @ 11-13 weeks

β HCG \Rightarrow ↑
 PAPP-A \Rightarrow ↓

(Pregnancy associated plasma protein A)

* USG for NT + Dual test
 ↓
Combined test*

2nd Trimester (27)

SOFT MARKERS (Not specific)
 ≥ 2 Soft Markers

↑ Risk of Aneuploidy
 15-20 weeks

Maternal Serum Marker

↳ do @
 15-22 wks.

→ Triple test

↳ hCG
 AFP

UE₃ (Unconjugated Estriol)

→ Quadruple test

↳ hCG

AFP

UE₃

+ Inhibin A (↑)

↳ Produced by placenta during 2nd & corpus luteum in Non-pregnant female

**

* Soft Markers \Rightarrow { Absent Nasal bone (Hypoplastic) \Rightarrow can be
 Most imp. \leftarrow { Nuchal fold thickness ↑, seen @ 1st
 ($\geq 6\text{mm}$) trimester at
 ↳ Screening ⊕ve

echogenic cardiac foci
 echogenic bowel foci

- Short femur
- Short humerus
- Short frontal lobe
- Short ear length
- Simian crease (single palmar crease)
- Short 4th middle phalanx (clinodactyly)
- Saddle gap
- Pyelectasis (Mild dilatation of Renal pelvis)

QQ

M/c Congenital cardiac Abnormality in Down's sx child

Endocardial cushion defect > VSD > ASD

→ Not central (More towards one side)

QQ

Gastroschisis is Not seen in Down's sx patient

Omphalocele → Covering Membrane (+)

↳ central

*

Confirmatory test

⇒

Karyotyping

M/c Method → "G Banding"

↓
In Metaphase

↓
Drug: Colchicine

1st Trimester

↓

Chorionic villous Sampling

⇒

≥ 10 weeks

M/c → 11-13 weeks

don't do before 9 weeks

↓

b/c it may cause

↓

Limb defects

- can result in False +ve test (Placenta)
secell (Late hair)
- Risky fetal loss 1%

- early Result + trophoblast (48-72 hr)

2nd trimester

↓

Amniocentesis

≥ 15 weeks

M/c = 16-18 weeks

don't do 11-14 weeks

↓

early amniocentesis

↳ fetal loss

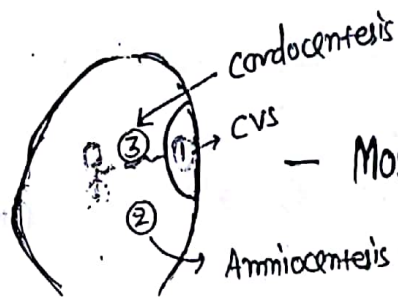
- More accurate
- More safe < 0.5%

- delayed (7-10 days) Result

↓

Amniocytes
Fibroblast

* Cordocentesis → Umbilical cord blood cells



— Most Risky ⇒ 3% fetal Loss.

from Umbilical vein (placenta end)

b/c it is stable end.

* Amniocentesis all USG guided procedure; Not blind - procedure.

* NIPT ^{costly} (Non-Invasive Pre-Natal test) : ⇒

- Genetic Material ⇒ cell free fetal DNA
- done on Maternal blood (Some amount of cell free DNA circulating in Maternal blood)
- ≥ 10 weeks
- this test is used as 2^o screening (costly)
- has to be followed by confirmatory test;
- takes 7-10 days in Results.
- tells us about Trisomy 13, 18, 21
Monosomy - XO

PHYSIOLOGICAL CHANGES AFTER PREGNANCY

Presumptive sign

Women experience

Reusell (eg \Rightarrow Amenorrhoea;
Morning sickness
etc)

Probable sign

Seen by doctors

Positive sign

Confirmatory

earliest = Δx = hcg ^{serum} / ^{urine}

USG

O/E \Rightarrow Fetal parts
 \hookrightarrow Fetal heart rate

Probable sign \Rightarrow i) Hegar's sign \Rightarrow Softening of Isthmus
(on Bimanual examination)
 \hookrightarrow Seen on 6th week

ii) Good sign \Rightarrow Softening of cervix
 \hookrightarrow Seen on 6th week

@ 8th weeks

iii) Chadwick's (Jaquimier's sign) \Rightarrow

Bluish discoloration of vagina/vulva

iv) Oslander's sign \Rightarrow Lateral vaginal fornix
Pulsations

v) Piskacek's sign \Rightarrow one half of uterus feels
more enlarged (Asymmetrical)

vi) Palmer's sign \Rightarrow Regular Rhythmic Uterine ⁽²⁹⁾ ⁵⁷ contraction
(No Pain; only felt by examiner)
⑧ 8th week

vii) Haltman's sign \Rightarrow Implantation bleeding (Not a problem)
⑧ 8th week

On USG \Rightarrow 1st sign of pregnancy on USG *

\Downarrow
Gestational Sac **

On TVS \Rightarrow 4-5 weeks (4 1/3 day)

empty bladder \leftarrow

On TAS \Rightarrow 5 weeks

Full bladder \leftarrow

TVS is \approx higher frequency & (25 Hz)

TAS is \approx Lower frequency (3 Hz).

Intradecidual sign \Rightarrow Marker of intra-uterine \odot
early

Yolk Sac appear \Rightarrow TVS = 5-5 week

TAS = 6 week

Fetal Pole \Rightarrow Cardiac activity $\begin{cases} \text{TVS} \Rightarrow 5-6 \text{ weeks} \\ \text{TAS} \Rightarrow 6-7 \text{ weeks} \end{cases}$

* Double-decidual sac sign \Rightarrow Marker of early intra-uterine \odot .

\swarrow Decidua capsularis \searrow Decidua parietalis

* Double bleb sign \Rightarrow Sign of early Intrauterine ♀

\swarrow \searrow
 Amnion Yolk sac

Q. Q. Earliest to see Gestational sac \Rightarrow 5th week on USG

Q. Q. Earliest time Gestational sac can be identified on TVS from LMP ??

Q. Q. Earliest time Gestational sac can be identified on TVS from fertilization ??

$$\hookrightarrow \text{LMP} - 15 \text{ days} = 30 - 15 = 15 \text{ days}$$

\Rightarrow What ever seen on TAS can be seen in TVS (vice versa is Not true)

\Rightarrow In Ectopic : Ring in Uterus \Rightarrow Single Ring in Uterus

\Downarrow
 "Pseudosac" \Rightarrow pr. in ~~center~~ uterus.

General changes \Rightarrow ① Additional Calorie Requirement

In ♀ ; everywhere Progesterone excret \Rightarrow Obstetric cholestasis } \Rightarrow estrogen
 salt & water Retention
 \uparrow

\Downarrow
 350 Kcal/day

\hookrightarrow In 1st trimester = No additional Calorie Requirement.

Brought about by
 \uparrow
 Retain salt/water

② Average wt. gain

\hookrightarrow 12 kg (10-14 kg)

③ BMR \Rightarrow \uparrow by 20%

④ salt/water \Rightarrow 6.5L

④ Plasma osmolality \Rightarrow ves (10 mosm/kg)

30

⑤ Plasma volume $\rightarrow \uparrow 40\%$

Red cell Mass $\rightarrow \uparrow 20\%$

Hemodilution condition \Rightarrow Anemia of Pregnancy

Anemia \Rightarrow Hb $< 11 \text{ gm\%}$; Hematocrit $< 33\%$

\hookrightarrow M/C = Iron deficiency Anemia*

Total Fe Requirement = 1000mg

300mg (Fetus)

Best test = S. Ferritin

Prophylaxis = GFA (100mg Fe + 500 Mg FA) \nearrow in Fetus form

\rightarrow 1 tab

\rightarrow 6 month during pregnancy

6 month after delivery

T/t of Anemia \Rightarrow IFA tab (2 tabs) \nearrow in Acute blood loss & Hg $< 6 \text{ gm\%}$

Parenteral

i) If the patient is Not compliant by oral tab

ii) Not tolerating oral tab

iii) Malabsorption sx.

Blood transfusion

i) If $> 34 \text{ weeks}$ @ & Hg $< 7 \text{ gm}$ (even No sign/symptom of Heart failure)

ii) If $< 34 \text{ weeks}$ @ & Hg $< 5 \text{ gm}$ (even No sign/symptom of Heart failure)

iii) Anytime sign & symptom of CHF

⑥ TLC \Rightarrow \uparrow (It doesn't Mean Infection)
 \hookrightarrow 15,000 during Pregnancy
25,000 after Postpartum.

⑦ DLC \Rightarrow Neutrophilia
ESR \uparrow
CRP \uparrow

⑧ Platelet count \Rightarrow Average platelet count \downarrow
 \hookrightarrow Not causes Thrombocytopenia

⑨ Clotting factors \Rightarrow all \uparrow except \Rightarrow Factor II & III.

⑩ Insulin Resistance \Rightarrow Hyperinsulinemia
 \uparrow as \uparrow \downarrow \uparrow
Significant > 24 weeks
 \rightarrow Fasting - Hypoglycemia
Post-prandial - Hyperglycemia

aa

* Anemia : $< 11 \text{ gm/dl}$

Severe Anemia : $< 7 \text{ gm/dl}$

Very severe Anemia : $< 4 \text{ gm/dl}$

CVS **

(31)

↳ Plasma Volume $\Rightarrow \uparrow 40\%$
 Red cell Mass $\Rightarrow \uparrow 20\%$
 Cardiac output $\Rightarrow \uparrow 40\%$

O_2 demand of tissue $= \uparrow 20\%$

feature of $\leftarrow O_2$ carrying capacity = ↓ *
 Hb; Not of
 Red cell Mass

A-V O_2 gradient of tissue = ↓

- All heart sounds are loud = Loud S₁
- S₃ (Gallop Rhythm)
- Systolic Murmur (Ejection systolic Murmur)
 ↳ Physiological up to grade 2.

Diastolic Murmur

↳ almost/ Always Pathological

- Heart Rate ↑ (by 16-18 beats/min above baseline)
 (<100)

- Split S₁

- Apex beat \Rightarrow heard @ 4th ICS (b/c heart is Rotated Anteriorly & Pushed up).

on CXR \Rightarrow cardiac silhouette (appears big)

cardiomegaly \Rightarrow always pathological

on ECG \Rightarrow LAD \Rightarrow Physiological
 \hookrightarrow Left Axis deviation **

- Blood pressure = DBP $>$ SBP \downarrow (Both Fall)
(10 mm Hg) (all vaso pressure \downarrow)

E/P (Estrogen / Progesterone)

- \rightarrow i) vasodilation
ii) Resistance against vasopress

beginning - 5 weeks (\downarrow in DBP begins @ 5 weeks)

Maxim = 24-26 weeks

after 26 weeks = beginning \uparrow
(Come back to pre-pregnancy value)

* ≥ 20 weeks \Rightarrow Supine hypotension syndrome

\Downarrow
Gravid uterus compresses IVC
 \swarrow
Changes position to Left Lateral position
 \hookrightarrow \uparrow uterine-placental circulation
Fetal O₂ saturation by 10%

* Preload $\Rightarrow \uparrow$

afterload $\Rightarrow \downarrow$ (d/t fall in systemic vascular resistance)

* Ejection fraction = No change

Central venous pressure = No change

(32)

↓
Persistently distended Neck vein

↓
Pathological Always *

CHF (Highest Risk)

↓
cardiac output Tes seen

Immediate > 2nd stage of Labour > 32 weeks
Post-partum
(3rd stage)

→ Co is Not Tes until
it goes to 2nd stage of
Labour, after 32 week of
P.O.G.

Q9 Co is Max^m @ →

a) 28 week; b) 32 weeks; c) @ term; d) 36 weeks

Q9 Pre-eclampsia Not commonly dx in

→ 3rd trimester.

26/4/18

KIDNEY

→ Tes Renal blood flow by 80%.

GFR Tes by 50%.

→ S Creatinine ↓; BUN ↓

S Uric acid Level → No change b/c Reabsorption.

* S. Creatinine ↑ } Pre-eclampsia*
S. Uric acid ↑ }

* Kidney - enlarge by 1cm

* Hydroureter - b/c of Progesterone

↳ Smooth Muscle Relaxant

↳ R+ side > L+ side; why??

9m @ Uterus becomes dextrorotation towards R+ side

↳ Compresses Right Ureter

@ the Pelvic Brim.

but still doesn't become incon-tinent
as Urethral pressure also ↑

↓
Bladder Pressure ↑

↳ From 8cm H₂O

↳ TO 20cm H₂O.

* Urinary Stasis ⇔

Urine — Routine — Asymptomatic Bacturia > 10⁵
 — Microscopic

↓
Treat; 'if we don't treat then
chances of Pyelonephritis becomes high.

* Glycosuria - b/c Renal threshold ves.

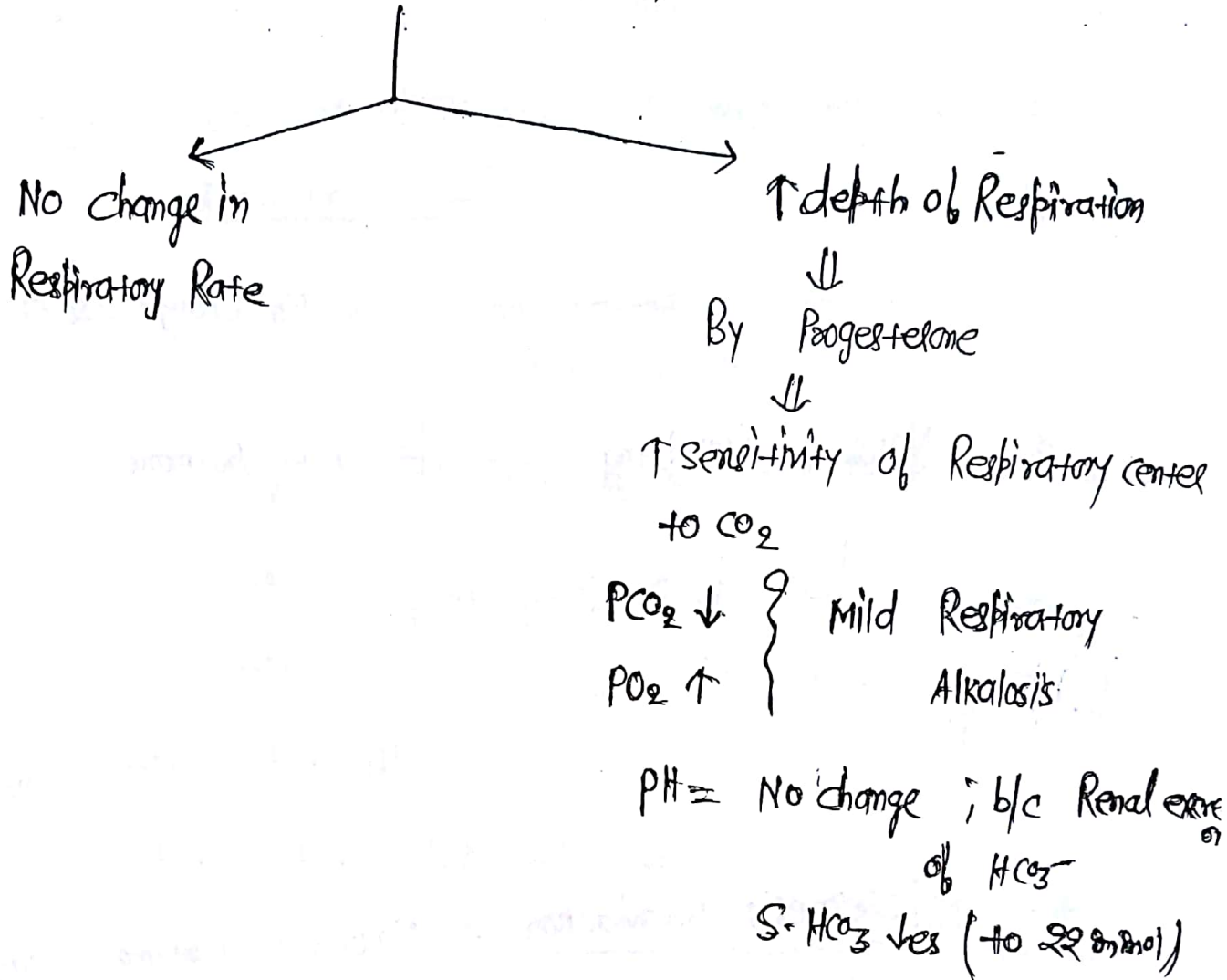
* Proteinuria - ≥ 0.3 gm/24 hr of Urine sample

↳ Not Physiological; It's Pathological

Respiratory system

(33)

- * Tidal volume \uparrow
- * Minute ventilation \uparrow (40%)



- * Diaphragm Rises by 4cm

↑ transverse thoracic diameter = 2cm

∴ Total Lung Capacity = ↓

∴ Vital capacity = No change

so, ERV ↓
RV ↓

IRV = No change

pH — RR (Respiratory Rate)
— Fev₁
— P_{awp}

* Diaphragmatic Excursions \Rightarrow Tex in Pregnancy.

GIT

* GERD Seen \Rightarrow b/c LES tone less by Progesterone
& Gastric Pressure Tex.

* Gastric emptying time shows No change in all trimesters.

* Nausea, vomiting = d/t hCG hormone

\rightarrow 1st line drug in ϕ = Pyridoxine

\downarrow No Response

Doxylamine + Pyridoxine

* Hyperemesis Gravidarum \Rightarrow Nausea & vomiting causes
either $\geq 5\%$ wt. loss
&/or
Ketonaemia

\rightarrow d/t hCG
 \rightarrow Infection of H. Pylori

Seen in Multifetal ϕ ; & female sex fetus;

* 2 vit. deficiency \rightarrow vit. K - Coagulation defect
 \rightarrow vit. B₁ - Wernicke's encephalopathy,

- Can cause Esophageal tear (Mallory-Weiss tear);
- Renal failure - (Acute Renal failure). (34)

Rx \rightarrow Stop all oral Intake
give ivr Fluids + ivr Antiemetics.

* Liver \Rightarrow ALP \approx Gross Tes
 \hookrightarrow b/c Placental - ALP
 \rightarrow Not a Marker of cholestasis (it is Physiological Tes)

- Tes Production of Albumin/Globulin
- S. proteins \rightarrow Fall (b/c Production is less than Volume Tes)
- AST, ALT both \downarrow es

Endocrine system*

Pituitary \rightarrow N In size 135x

b/c Lactotroph TT (vascular supply TT)

In severe PPH — Vascular supply — \downarrow to Pituitary

\downarrow
Infection

\downarrow
"Sheehan's syndrome"

Sheehan's Sx \Rightarrow M/c Presentation \Rightarrow Failure to Lactate

2nd M/c \Rightarrow Amenorrhoea

Usually - Ant. Pituitary affected

\Downarrow
Post. Pituitary spared.

\Rightarrow if a ♀ doesn't Lactate - Menses by 6-8 weeks.

S. Prolactin Level - Highest

Pregnancy

Lactation

\Downarrow
After delivery S. Prolactin ↓ by 50%

Prolactin - Milk Synthesizing hormone

Oxytocin - Milk Letdown / Ejection hormone

1st Stimulus \Rightarrow Initiation of Lactation
($\downarrow \bar{E} + \bar{P}$)

Fall of E & P also causes \Rightarrow Post-partum depression
(Blues)

THYROID \Rightarrow • Thyroid binding globulin \uparrow (Estrogen) ^{d/t} ³⁵ ⁶⁰

• Total T_3 & T_4 \uparrow } \Rightarrow \uparrow Production from the gland

• Free T_3 T_4 \uparrow (slightly) } \downarrow

• TSH \downarrow (slightly) Why? \Rightarrow b/c of hCG

• I_2 Requirement \uparrow \downarrow

(♀ & Lactation) both have RDA = 250 μ g/day, $\alpha =$ TSH (same as TSH)

Pregnancy is Euthyroid condition

• I_2 excretion test.

* M/c of hypothyroidism in ♀ \Rightarrow Hashimoto's ds

M/c of hyperthyroidism in ♀ \Rightarrow Graves ds.

Q K/c/o hypothyroidism ; L. thyroxine 25 μ g

Dx = Pregnancy \Rightarrow Test by 50% (b/c some part of thyroid doses become degenerated)

Hypothyroidism may cause abortion.

Maternal Nerve Injury \Rightarrow M/c in Lithotomy position

\downarrow

Common Peroneal Nerve

* M/c in Postpartum / Peripartum / Intrapartum

\downarrow

Lateral cutaneous N. of thigh > Femoral N. extended Lithotomy position

* Foot drop in ♀ is d/t "Lumbosacral Plexus Compression."

- *

Fetal Swallowing \Rightarrow 10 weeks

Fetal breathing Movement \Rightarrow 11 weeks

Fetal Urine production \Rightarrow 12 weeks

Fetal Meconium production \Rightarrow 16 weeks

IgM production in baby \Rightarrow 20 weeks

by mother \leftarrow IgG transfer in baby \Rightarrow 16 weeks

Surfactant synthesis begins \Rightarrow 20 weeks

Surfactant appears in Amniotic Fluid \Rightarrow 28 weeks

Glucagon production \Rightarrow 8 weeks

Insulin production \Rightarrow 12 weeks

H-P (circulation) \Rightarrow 12 weeks*

AMNIOTIC FLUID

(36)

Major Source \Rightarrow Fetal Urine

Major Removal \Rightarrow Fetal Swallowing

Major Source \rightarrow in 1st 12 weeks \Rightarrow Ultrafiltrate of Maternal Plasma.

In 12-20 weeks \Rightarrow Transudate across fetal skin

in >20 weeks \Rightarrow Urine.

* 98% Amniotic fluid \Rightarrow Water (Nutrition)
 \rightarrow Not help in Nutrition⁹⁹.

* Colour of Amniotic fluid \Rightarrow Straw coloured

Green colour = Meconium-stained

Dark = Abrasion

Golden = Rh Incompatibility

Tobacco Juice (dark brown) = GUD

Greenish Yellow (saffron) = Post Maturity

* pH = 7-7.5

* Osmolality = 260 mOsm/l.

* Water is Replaced in every 3 hrs.

Normal

AFI (Amniotic Fluid Index)
5-24cm

DVP (Deep Vertical Pocket)
2-8cm

Polyhydramnios

≥ 25

≥ 8

Oligohydramnios

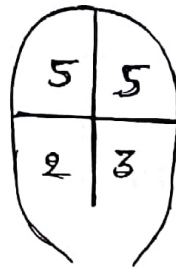
≤ 5

≤ 2

More Common Method

Better (No quadrants so less error)

AFI \Rightarrow



\Rightarrow divide in 4 quadrants Arbitrarily

15 = Amniotic fluid

DVP \rightarrow Not divided in quadrants; only Pocket division.

* Absolute value \Rightarrow Polyhydramnios = 2L

Oligohydramnios \approx 200ml

* In twin ϕ ; we can't use AFI; only use DVP;

\hookrightarrow b/c we don't know about quadrants of both fetuses.

(37)

Mild — Poly. — Idiopathic
— oligo.

Moderate — Severe — Poly. — Gross Congenital Anomaly
— oligo.

GUT > NTD

Leakage of CSF in Amniotic Fluid

b/c of Fetal Swallowing Problem

duodenal Atresia

double bubble sign.

B/L Renal Agenesis

Posterior Urethral valve

"Key hole sign" on USG seen.

Urethra

Bladder (dilated)

Lady comes with Poly / oligo (Moderate-severe)

diabetes

Leaking Amniotic fluid

seen by Per speculum examination.

* Polyhydramnios have high Risk of → (Moderate - severe)

- ① Maternal Respiratory distress;
- ② Pre-term Labour - < 37 weeks
- ③ Premature Rupture of Membrane - Membrane Rupture before Labour.

Pre-term premature Rupture of Membrane - < 37 weeks & PROM.

- ④ Abruptio - Separation of Placenta from Underlying decidua.

↳ prevented by Controlled ARM

↓
Multiple pin prick holes into the Membrane

- ⑤ Cord prolapse

↳ obstetric emergency b/c of temperature change to → vasospasm
Cord

↳ severe fetal distress.

↓
do emergency C.S.

- i) Reposit the cord above the presenting part;
- ii) Fill the bladder;
- iii) elevate the buttock;
- iv) or by MCK's or IV fluids given.

↳ } Cord pulsation (Absent)
Fetal heart Rate (Absent)
→ do vaginal delivery..
(GUD case)

- ⑥ Malpresentation ;
- ⑦ PPH ;
- ⑧ GCM ;
- ⑨ Diabetes ;
- ⑩ Amniotic fluid embolism

* Oligohydraminos have high Risk of \Rightarrow

- ① Pulmonary hypoplasia;
- ② Fetal distress (cord compression)
- ③ Malpresentation
- ④ GUGR
- ⑤ PE (Pre-eclampsia) ^a association \bar{c} oligo. b/c of Uteroplacental Insufficiency (UPI)
- ⑥ Early in pregnancy oligo.
↳ viral Infection (TORCH/zika)

⑦

Amniotic band Sequence \Rightarrow

tears in Amniotic Membrane



Severe oligohydramnios (d/t leakage)



Bands & tight wrap around fetus.

M/C Anomaly in Amniotic band sequence



Limb Anomalies > Craniofacial Anomalies

⑧

Amnion Nodulum



S. oligo + yellowish Nodules on the Membrane
↳ severe

⑨

Compression defect \Rightarrow CTEV (Club foot)

Q9

Diagnostic Amniocentesis \Rightarrow ① Karyotyping;

② Neural tube defect \rightarrow AchE / AFP*

Best screening test for NTD \Rightarrow USG

Best test for NTD \Rightarrow AchE / AFP (Amniocentesis)

Other screening test for NTD \Rightarrow Serum Alpha-fetoprotein
16-18 WK

> 9-10 WK (available at maternal)

* AFP Peak $\begin{cases} \text{In Fetus} - \underline{13 \text{ weeks}} \\ \text{In Mother} - \underline{32 \text{ weeks}} \end{cases}$

$\hookrightarrow t_{1/2} = 5-7 \text{ days.}$

(39)

* \downarrow Level of AFP seen in \Rightarrow Down's Sx;
Diabetes;
obesity;
Molar \otimes ;
GUD.

③ Lung Maturity \Rightarrow M/C = L/S Ratio $\Rightarrow 72 \Rightarrow$ Mature

\hookrightarrow Best test \Rightarrow PG (Phosphatidyl Glycerol)

\hookrightarrow Not affected by presence of
contaminants.

\rightarrow in Diabetes Mothers.

④ Hemolytic Anemia \Rightarrow In Fetus

⑤ Asks of acute viral Infection in fetus

\hookrightarrow Amniotic fluid Polymerase chain reaction. 99

In Acute Maternal Infection



4 fold Rise in Ab titre in Paired sera.

— Avidity test.

⑥ Chorioamnionitis

*

<u>P.O.G.</u> -	<u>Amount of Amniotic Fluid</u>
12 WK →	50mL
16 WK →	250mL
20 WK →	400mL
32 WK →	1 Litre (Max ^m)
36 WK →	900mL
Term 40 WK →	800mL
42 WK →	200mL

POST-PARTUM HEMORRHAGE

(40)

3rd Stage \Rightarrow Placenta expulsion

\hookrightarrow Avg. time - 15-20 min

Prolonged > 30 min.

Signs \Rightarrow ① Gush of blood;

Best sign \leftarrow ② Lengthening of cord; Apparent
 ③ Subpubic bulge; Permanent
 ④ Fundal height test

Best sign \Rightarrow Placenta lying in vagina $>$ Lengthening of cord

⑤ Avg. Blood Loss \Rightarrow Vaginal delivery = 500ml
 C.S. = 1000ml
 Twin vaginal delivery = 1000ml

PPH

1°

\leq in 24 hrs of deliv

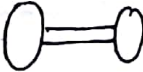
2°

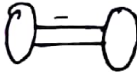
> 24 hr upto 12 weeks

M/c cause \Rightarrow Uterine Atony

Retained Placental tissue

Placental Abnormality which is RIF for PPH \Rightarrow


"Succenturiata"


"Bilobata".

4 Ts — Tone (Uterine Atony)

Trauma

Tissue (Retained Placental tissue)

Thrombosis (defect)

* R/F for Atonicity \Rightarrow \Rightarrow Multifetal \odot

ii> Macrosomia

iii> Polyhydramnios

iv> Induction of Labour

v> Augmentation of Labour

vi> Precipitate Labour

(onset - expulsion \leq in 3 hours)

vii> Any kind of APH

viii> Multiparity

ix> Diabetes Mellitus

x> Pre-eclampsia

xi> Chorio amnionitis

* Most Important things for placental separation

\hookrightarrow Uterine contraction.

Plane of Placental separation ⇒

Methods by which placenta separates ⇒



Placental separation

↳ Starts from @ centre

Schultz Method*

• External — after complete separation
bleeding

• Blood loss ↓ (Total)

• Side presents @ vulva = fetal side

Shiny side
↳ Schultz*
80% cases (M/c separation)

• Retroplacental clot or
is formed

* M/c of PPH ⇒ Atomy > Genital tract Trauma > Retained tissue >
Inversion > Rupture Uterus > Amniotic fluid embolism.

** 80
Spongiosum

(41)

↳ Layer of decidua*



Starts @ Periphery

Duncan's Method*

begins @ onset of
separation

Total blood loss ↑

Maternal side



Dirty

↳ Duncan's Method*

20% cases

• Absence of Retroplacental
clot.

Q9

Prevention of PPH (AMTSL)

↳ by WHO

↳ Active Management of Third stage of Labour.

Components ⇒

1) Give uterotonic Agent Immediately after delivery
↓
c/in 1 min.

a) Doc! Oxytocin

MOA ⇒ by Tes
Intracellular cat²

• Oxytocin causes
Release of Pinf²
from decidua.

↓
10 IV

10m bolus onset duration
c/in 3 min 3 hr

ilv - infusion Immediate 1 hr.

↳ Not give bolus b/c it is

R/F for = Hypotension

↓

Reflux Tachy. | Arrhythmia | MI | Cardiac Arrest Q9

- Naturally synthesis oxytocin ⇒ Nonapeptide

Artificially synthesis oxytocin ⇒ Octapeptide

- Synthesized from - hypothalamus

↓

Paraventricular Nucleus

- $t_{1/2} = 3 \text{ min (3-5 min)}$

- Stored in cold chain ($2-8^{\circ}\text{C}$)

↓

b) Methergin (Ergometrine)

(42)

↳ 0.2 mg i/m

↓
don't give i/v → causes transient severe hypertension

So, c/i'm ⇒ Pre-eclampsia

Eclampsia

CVS disease

Peripheral vascular disease

tetanic contraction (Acts more on LUS; while oxytocin on all uterus).

Brown colour - b/c of photosensitive nature.

c) Syntometrine (5 U oxytocin + 0.5 mg Methergin)

• Very Potent

• Not Doc → expensive
→ Not availabled) Carbetocin = Synthetic oxytocinOctapeptide / Longer t_{1/2}

↳ 100 µg slow i/v over 1 min.



e> Misoprostol = PGE₁ analogue



Prophylaxis = 600mg (per oral)

Route = oral (In India ⇒ ~~per~~ Rectal)

Asthma is Not a C/I.

M/c side effects ⇒ ^{**}Hyperpyrexia (fever + chills).

↳ directly proportional to dose

Other side effects ⇒ Nausea; Vomiting; Abd. Pain; Hypotension.

II> Delivery of placenta by controlled cord traction!

eg. Klaus "Modified brandt andrew Method"

Rt. hand ⇒ Hold cord ↳ do only when trained birth staff present.
Lt. hand ⇒ Push Fundus up.

III> Delayed cord clamping ⇒

↓
≥ 60 Sec. (1-3 min)

↳ Goes More blood to fetus (80ml)

↑ Hb by 2gmy. ← 50mg Fe

In HIV patient ⇒ Delay cord clamping done
transmission happen during Labour.

Never done due to
AMTSL in Normal Labour.
early cord clamping

↓
C in 60sec

↳ Indications ⇒

- Baby Needs Resuscitation
- Rh Incompatibility
- Baby is known case of Heart disease

(43)

IV) Intermittent Uterine tone assessment ⇒

Uterine Massage = Not done

Q. Most Imp. component ?? ↳ Not a component of AMTSL.
 (A) I; (B) II; (C) III; (D) IV ↳ 1st component is Uterine tone.

Management of PPH : $\left(\text{Shock Index} = \frac{\text{Heart Rate}}{\text{SBP}} \right)$

↳ (N) = 0.5 - 0.7

if > 0.9 ⇒ Immediate Resuscitation

Specific T/t (Algorithm)

Bimanual

↳ B/M Uterine Massage

+

call for help

+

Uterotonics given

↳ doc P/t

↳ oxytocin

↓

20IU — 40IU / 500ml of

NS / RL

Not in 5+ dext.

↓

If we give Large doses

Large duration

electrolyte deficient medly

↓

Result in water intoxication

Symptomatic T/t

i) 2 Large bore ilv

Cannula

(14/16 Gauge)

ii) ilv fluids - crystalloids

iii) Arrange blood & blood products;

iv) Catheterize - Urine output

v) Blood group / Rh / CBC / Coagulation Profile

↓

to decrease Morbidity & Mortality Rate.

↓ if Not Responding

Inf. Tranexa (Tranexamic Acid)

↓

↳ Anti fibrinolytic drugs

Antifibrinoly

1gm slow

↓

if Not Responding

Methergin
0.2 mg (i/m)

carboprost (Methy/ Pifex)

↓

0.25 mg (i/m)

↳ H/O Asthma ⇒ Not given; Pt. dies d/t bronchoconstriction

↓

Dianthoa (M/c side effect
if Not Responding. of carboprost)

Misoprostol → to be used ^{when} i/m; i/v can't be given.

↳ Ht = 800mcg (Sublingual)

Not given P/V (Per vagina)

can give P/R (Per Rectal)

* All drugs total to happen upto 30min.

↓ if Not Responding.

(44)

Placenta - completeness

Genital Tract trauma

Uterus feel Like Intermittent atonicity

PPH \Rightarrow Uterus tonically contracted then Reason is

- i.e Not tonically contracted b/c of Retain placental tissue

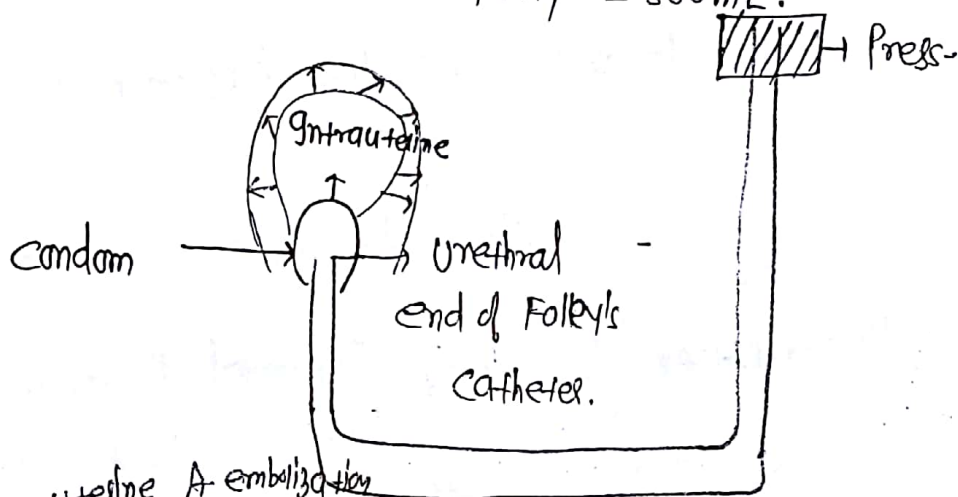
No trauma
Placenta complete

Balloon Tamponade

Bakri Balloon tamponade

Sengstaken Blakemore

Maxm Fluid capacity = 500 mL.



uterine A embolization
do UAE \Leftarrow Minimally Invasive Method if Not Responding
do in hemodynamically stable Pt. Surgical method
Availability.

Surgical Management

- Uterine compression suture (B-Lynch suture) ^{→ Brace}



↓
Applied on uterus & helpful
only in Atonic PPH



if Not Responding

B/L Uterine A. Ligation

↓ if Not Responding

B/L - Anterior division of Internal iliac

↳ 5 cm distal to bifurcation of common iliac
↳ It ves Pelvic Pulse pressure by 80%



if Not Responding

Hysterectomy (Possible $\frac{+}{-}$ Sub total Hysterectomy
(Remove uterus - spares the
K/oa "Supracervical Cervix
Hysterectomy")



if bleeding
continues thinks
about "D.I.C."

G-GU.

↳ DIC **

↳ to Improve sexual life of
female

TRAUMA → Perineal tear



to prevent

- ① do Routine episiotomy ⇒ No
- ② one hand = Support the perineum
- ③ other hand = Maintain flexion of head
- ④ tell the Mother Not to push @ the time of head delivery.
- ⑤ NICE ⇒ application of warm Guidelines Perineal compresses

differs from episiotomy ⁽⁴⁵⁾



It is surgically planned Incision.

done in special cases



Forceps; Breech condⁿ

MedioLateral

Median



• ↑ Pain

extends

• ↑ Dyspareunia



• breaks down easily

Rectum sphincter & Mucosa

• ↑ Blood loss

• Poor Cosmetics

degree of Perineal tear ⇒

Repairable In Labour Room.

1st degree — vaginal Mucosa & Skin

2nd degree — Perineal Muscle

3rd degree — A — < 50% EAS torn

B — > 50% EAS torn

C — Both IAS & EAS torn

complete Perineal tears

4th degree — upto Rectal Mucosa

Complete Perineal tears \Rightarrow obstetric emergencies
 \approx In 24 hrs
 \hookrightarrow 3 weeks

1st to be Rejoined \Rightarrow Rectal Mucosa

Sphincter \Rightarrow End-End
Anastomosis

Mucosa = Continuous suture

Muscle = Interrupted suture

Skin = Matthews suture

\downarrow
Internal Anal sphincter (IAS)

\downarrow
External Anal sphincter (EAS)

\downarrow
Vaginal Mucosa

\downarrow
Muscle

\downarrow
Skin

Episiotomy

M/c used suture = Vicryl (Polygalactin-910)

* Another Tear \Rightarrow Hematoma

\hookrightarrow M/c Presentation = Pain

Inability to pass
Urine

\uparrow H = Analgesics

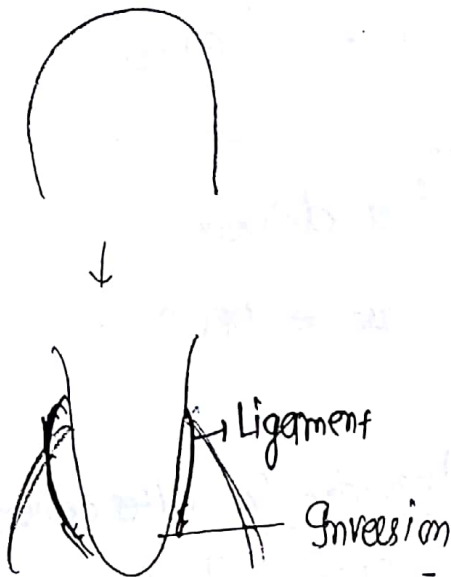
Monitor

ice-brake (Local application)

\rightarrow vasoconstriction

- Looks like bluish tender swelling
- Surgical Mx =
 - ① Shock
 - ② expanding in size
 - ③ excruciating Pain (hematoma expanding internally)
↳ Muscle.

* In Inversion ⇒ Stretch on the Ligament



⇓
Neurogenic Shock (1st shock to develop)

↳ Hemorrhagic shock

↳ death.

Rx ⇒ do Manual Repositioning
(Johnson's Method)

Part which comes out Last;
has to be Reposited first.

↓ if fails

Hydrostatic Method (O'Sullivan Method)

↓ if fails

Surgical management

Surgical Management
for Inversion

Haultain
Huntington
Spinelli

- emergency; give general Anesthesia; Not spinal Anesthesia



Relaxes uterus

also in Inversion

Manual Removal of Placenta

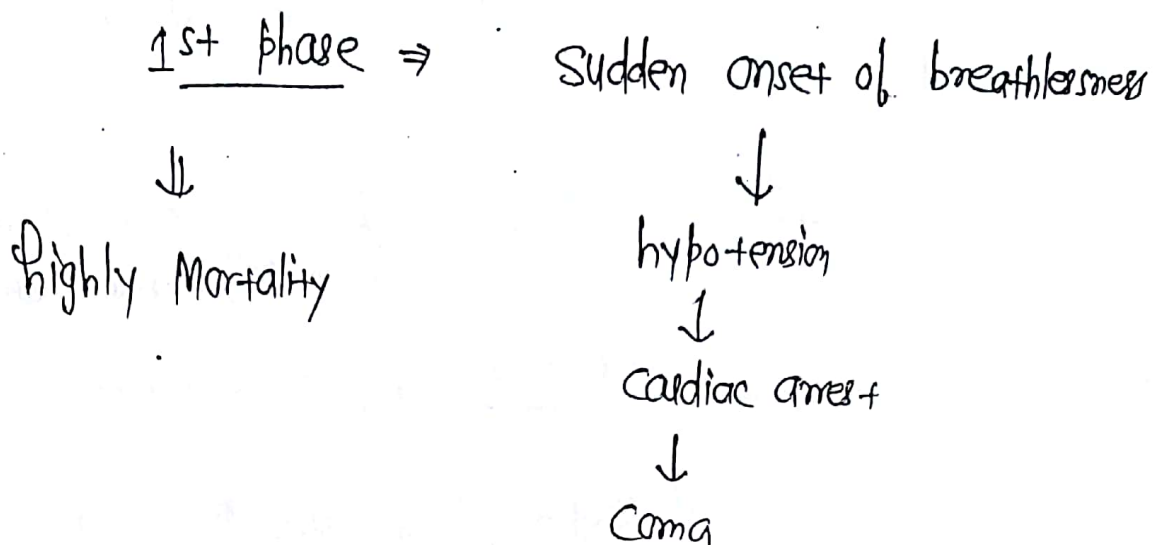
Hemodynamically Unstable patient

QA. Pt. goes into shock ^{or death} after delivery
Most probable cause \Rightarrow PPH

QA Pt. goes into shock immediately after delivery
- Most probable cause \Rightarrow Inversion

QA Pt. goes into shock (unexplained) after delivery
Most probable cause \Rightarrow Amniotic fluid embolism
(Very Rare / diagnosis of exclusion)
Klas "Anaphylactoid syndrome of pregnancy"

* clinical diagnosis of AFE \Rightarrow No Lab test to confirm or Refute diagnosis. (47)



2nd phase \Rightarrow Pt. goes into DIC + Hemorrhage

\Downarrow

death seen

Perf we can do \Rightarrow Sample from Pulmonary vessels

\Downarrow

Lamugo / vernix / amniotic fluid seen

DIC

M/cc = - Abruption

also by = AFE

Massive Hemorrhage (APH/PPH)

Sepsis (septic abortion)

and (≥ 4 weeks risk of DIC)

Management of GUD \Rightarrow wait & watch



Must go into spont. Labour \approx in 2 weeks

\hookrightarrow signs \Rightarrow Robert's sign \Rightarrow Comes @ 12 days
 \hookrightarrow gas in Major blood vessels

Spalding sign \Rightarrow Comes @ 7 days

Ball sign \Rightarrow Hyperflexion of spine

Buddha \Rightarrow Subcutaneous edema

sign

\hookrightarrow seen in hydrops fetalis

\hookrightarrow Not a sign of GUD.

PRE ECLAMPSIA

(48)

* Gestational HTN \Rightarrow i) BP $\geq 140/90$ on 2 occasions;
4-6 hr apart

ii) BP high > 20 weeks;

iii) Return to (N) \bar{c} in 12 weeks Post-partum.
iv) No evidence of Proteinuria

* Blood Pressure should taken in sitting position.

* 5th Korotkoff sound heard

* Pre-eclampsia \Rightarrow i) BP $\geq 140/90$ on 2 occasions; 4-6 hr apart

ii) BP high > 20 weeks

\bar{c} Any of the following

Proteinuria

$\geq 0.3 \text{ gm} / 300 \text{ mg}$

in a 24 hr Urine Sample

or

Urinary protein ≥ 0.3
creatinine

OR

Dipstick (1+)

End-organ damage

(It could be Any of the following).

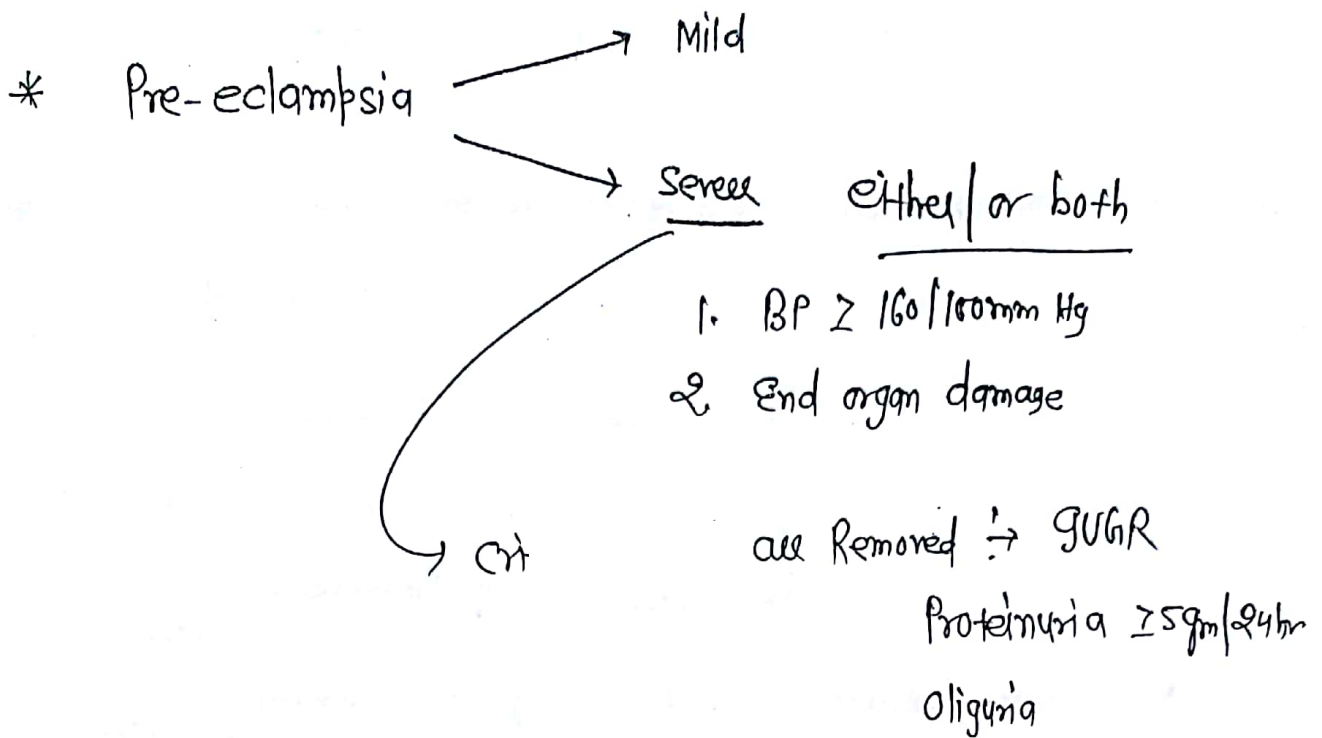
i) Platelet count $< 1 \text{ Lakh}$;

ii) Serum creatinine > 1.1

iii) Liver enzymes all More than
twice the (N) value

iv) Pulmonary edema

v) cerebral / visual symptoms



* Eclampsia \Rightarrow Pre-eclampsia + Seizures

* Chronic HTN in ♀ \Rightarrow i) BP is high before conception;
 ii) BP is high in 1st 20wks;
 iii) BP Remaining high $>$ 12 week post partum period.

* Chronic HTN \pm Superimposed Pre-eclampsia \Rightarrow

- i) New onset proteinuria beyond 20 weeks.
- ii) End organ damage $>$ 20 weeks
- iii) Uncontrolled HTN $>$ 20 weeks

- Impending eclampsia \Rightarrow if she has Any of the following sign & symptom \Rightarrow in epigastric pain (49)

\hookrightarrow Stretching of Liver

Capsule.

Spontaneous subcapsular hematoma

ii) Headache/dizziness

\hookrightarrow cerebral hypoxia

iii)

Blurring/diplopia/Blindness

~~Blindness~~ \rightarrow Central \rightarrow ~~capit~~ Lobe hypoxia

Scotoma

Peripheral = Retinal detachment

\hookrightarrow Hypertensive Retinopathy

iv) HELLP Syndrome

\hookrightarrow Mostly - Feature of severe Pre-eclampsia

\hookrightarrow Blood pressure in 15% of patient is (N)

Criteria \Rightarrow { H = Hemolysis (on Peripheral Smear
 \hookrightarrow Schistocytes (Helmet cells);
 \hookrightarrow S. Bilirubin ≥ 12
 E \Rightarrow Elevated Liver (ALT ≥ 70 IU)
 enzyme
 L \Rightarrow Low Platelet count (Plt. count $< 100,000$)

all should be \Leftarrow
 in patient

* M/c Presentation \Rightarrow Pain \rightarrow Epigastric Pain
(Rt. upper quadrant)

\downarrow
Seen in 3rd trimester

D/D \Rightarrow ~~Acute Fatty Liver of~~ \odot (close to HELLP)
of HELLP Sx
 i) Hepatitis
 ii) obstetric cholestasis

\swarrow
differentiated by
presence of

- ① Hypoglycemia
- ② Hepato Renal Sx
- ③ Coagulation defect
- ④ Pancreatitis

Pathophysiology \Rightarrow i) ABN of β -oxidation of Fatty acids \leftarrow Mother
(Mitochondrial)

Mainly in 3rd trimester

(More severe form
Liver injury) \rightarrow ii) LCHAD enzyme deficiency \leftarrow Fetus

by Postmortem
Liver Biopsy confirmed.

M/c Cause of Acute Liver failure in \odot = AFLP

- High Mortality Rate

* Acute hepatitis

↳ have Prodromal symptom.

(50)

- Liver enzyme Raised

- Bilirubin - Markedly Raised

M/c Acute hepatitis in ♀ ⇒ Hepatitis E

↳ high Mortality Rate

* Obstetric Cholestasis

- M/c Symptom ⇒ Pruritis

- seen in 3rd trimester

- Estrogen

- Mutation in Genes ABCB4
ABCB11

- Serum bile acids become accumulating

- ↳ diagnostic test

- IT ⇒ Urso deoxycholic Acid

↳ Most Risky for Fetus = Pre-term Labour;

Irritate the bile acids; ⇐ Sudden still birth;

Result in the all of
three.

Meconium Aspiration syndrome

Termination of \odot \Rightarrow (≡) 37 weeks \rightarrow (≡) 38 weeks
In obstetric cholestasis

* * Recurrence Risk of HELLP = 4-7%.

Obstetric cholestasis = 70%.

* Pathophysiology of Pre-eclampsia \rightarrow

Vascular Remodelling

Absent

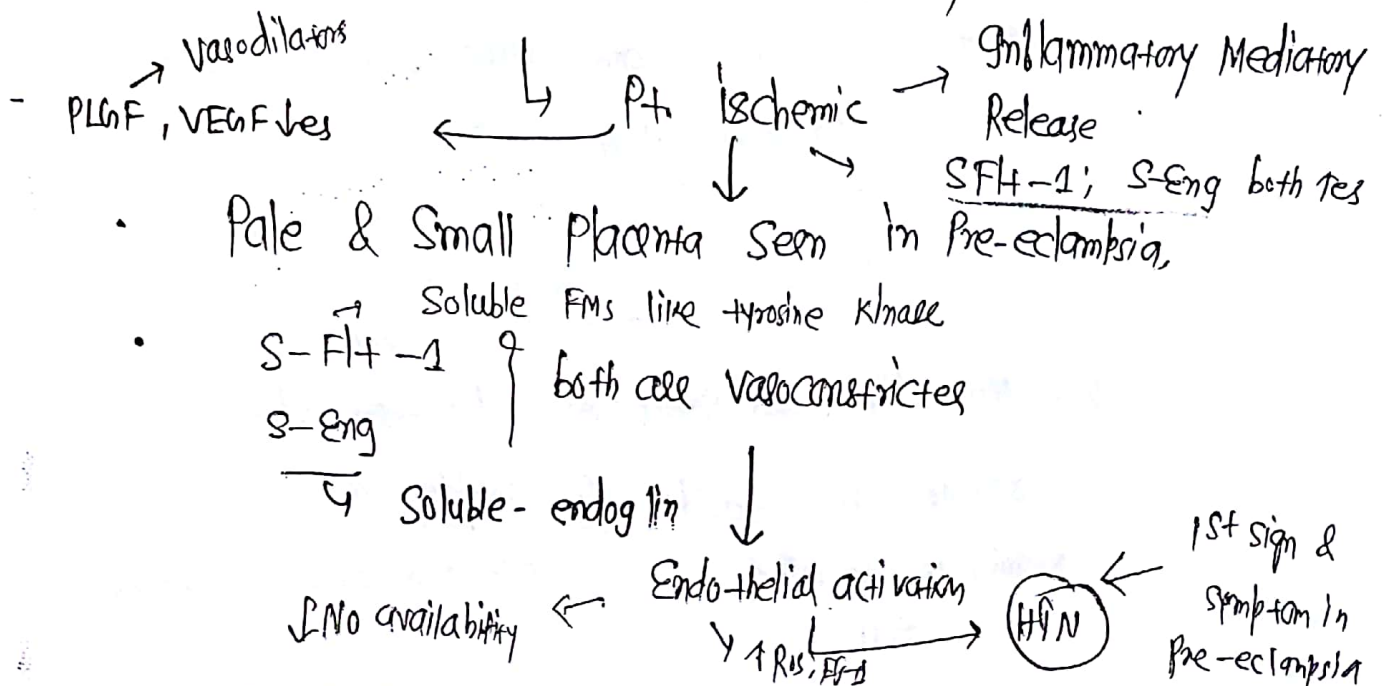


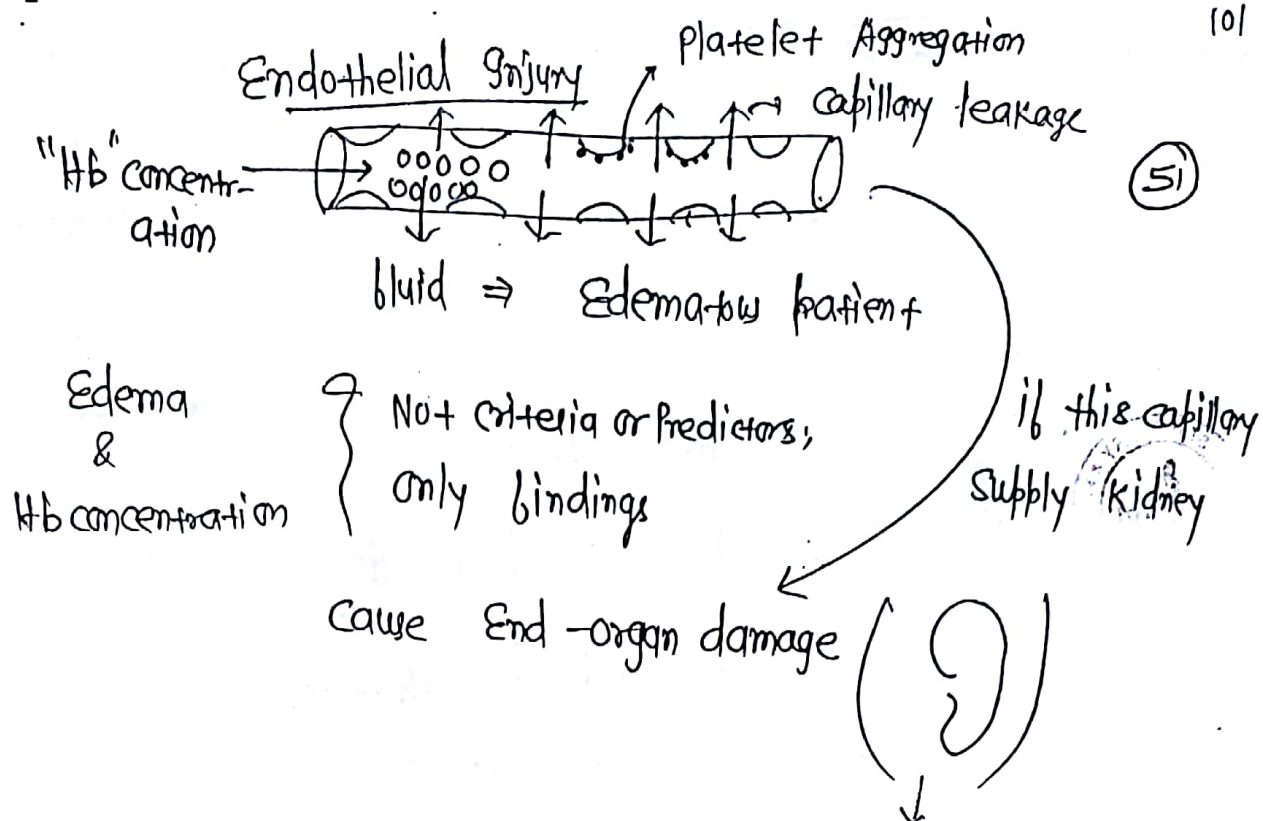
Vessels in Decidua Basalis



\hookrightarrow it means remain sensitive to vasopressin

Result in Utero Placental Insufficiency





1st & M/c organ affected in Pre-eclampsia

Kidney Shows "Glomerular Endotheliosis" on HPE.

\uparrow Serum Uric acid \Rightarrow also a ~~criteria~~ ^{binding} not criteria

* Immune theory \Rightarrow

Normally $\Rightarrow \downarrow$ TH₁ $\longrightarrow \uparrow$ TH₂ Response

In pre-eclampsia - this shift doesn't happen

\Downarrow
So, \uparrow TH₁

\Downarrow
Altered Immune Response to paternally derived Antigen.

Risk factor

- i> Primigravida; (b/c 1st time expose to Antigen)
- ii> Molar pregnancy (Extra paternal chromosome)
 - ↳ can develop early onset pre-eclampsia;
- iii> APLA
- iv> Multifetal @
- v> Chronic Hypertension
- vi> Renal disease;
- vii> Diabetes Mellitus;
- viii> obesity
- ix> extremes of age < 18; > 35 yrs
- x> Previous H/o Pre-eclampsia

Protective factor

- i> Smoking
- ii> It has \ominus ve association \bar{c} Placenta previa;
- It has \oplus ve association \bar{c} Abruption.

Predictors of Pre-eclampsia

- i> \uparrow sFlt-1
- ii> \uparrow s-eng
- iii> \downarrow PLGF
- iv> \downarrow VEGF
- v> \downarrow Urinary cat2 excretion,
- vi> \downarrow Urinary cat2 excretion,
- vii> \downarrow Urinary cat2 excretion,
- viii> \downarrow Urinary cat2 excretion,
- ix> \downarrow Urinary cat2 excretion,
- x> \downarrow Urinary cat2 excretion,

* Uterine A. Doppler

(52)

(N) → Notching — disappear by 22 weeks

Persistent + Notching — beyond 22 wk — Predictor for Pre-eclampsia

Prevention of Pre-eclampsia ⇒ • Aspirin

↓
150 mg — once in a day
— start in 12th wk
↳ Continue throughout 2nd
↳ only indicated for high Risk
Patient for Pre-eclampsia

• Ca Supplementation

↳ Prevent Pre-eclampsia only in women
who have Ca²⁺ deficient

- Salt Restriction
- Omega 3 Fatty acids
- Vit. C, E

} No Role in prevention.

* Management of Pre-eclampsia \Rightarrow

Symptomatic

- Anti-hypertensive
- Anti-seizure

Specific

- Termination of \varnothing

Stop progression

Reverse the damage

Anti hypertensive

i> When do you start Anti-hypertensive

L> if BD Persistently $\geq 150/100$ mm of Hg

ii> Which drug — Doc

L> Labetalol ($\alpha + \beta$ β -Blocker)

but; in Chronic Hypertension

L> Methyl dopa (safest Anti-hypertensive in Pre-eclampsia)

Doc for Acute HTN in \varnothing ($\geq 160/110$ mm of Hg)

L> acc. to ACOG, Any of these we as 1st line drug

① \swarrow
i/v Labetalol

② \downarrow
i/v hydralazine

③ \searrow
Sustained Release
Nifedipine

(53)

iv Labetalol



20 mg iv

↓ after 10 min

40 mg after 10 min → 80 mg → 80 mg upto total 300 mg/24 hr.

(4) iv Nitroglycerine ⇒ used in Pulmonary edema

(5) Lat + Resist + Refractory ⇒ Sodium Nitroprusside
 Anti hypertensive drugs cause (cyanide toxicity)

* Methyl dopa is Not given in Acute HTN
 ↳ b/c

ii> drugs not to be used as Antihypertensive →

i> ACEi ;

ii> ARBs ;

iii> Diuretics ;

iv> β Blockers ;

v> Diazoxide

iv) Target Blood Pressure \Rightarrow

Systolic blood pressure = 120-130 mm of Hg

Diastolic blood pressure = 80-90 mm of Hg

TERMINATION OF PREGNANCY

Gestational Hypertension / Mild Pre-eclampsia ; well controlled BP

\hookrightarrow @ 37 weeks

Severe Pre-eclampsia — well controlled — 1st — @ 34 weeks

* Indication of Termination of ♀ Irrespective of Gestational age

i) Impending Eclampsia;

ii) Eclampsia

iii) HELLP Syndrome;

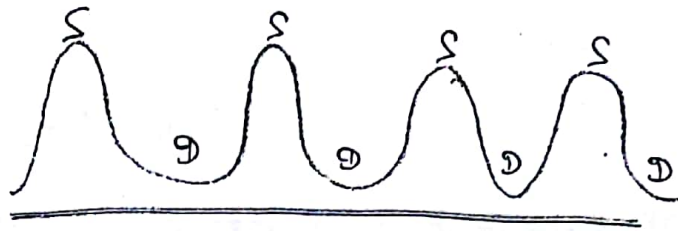
iv) Abruptio / Fetal distress

v) Uncontrolled HTN / Rising serum creatinine

vi) REDF — Reversal of End diastolic Flow
 \hookrightarrow doppler of Umbilical Artery.

① Umbilical Artery Flow $\Rightarrow \frac{S}{D}$ Ratio

(54)



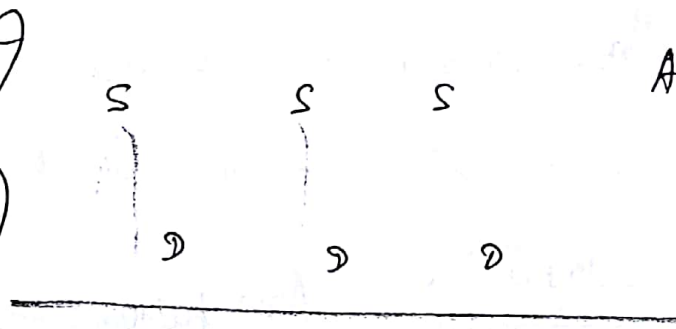
In ① Pregnancy $\Rightarrow \frac{S}{D}$ Ratio \downarrow as pos tes

In UPI $\Rightarrow \frac{S}{D}$ Ratio tes ($\geq 30 \frac{S}{D} \geq 3$)

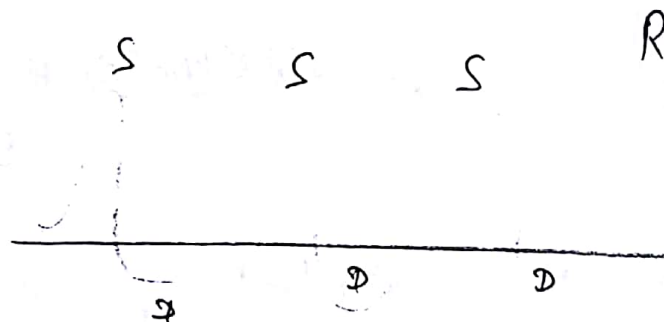
death \approx 48 hr.

- Omnidirectional condition

- Top irrespective of pos



Absent end diastolic flow



Reversed end diastolic flow

* Whenever possible do vaginal delivery \gg cesarean section
(High Risk)
Anesthetic complication
blood loss

*

In C.S.

↳ Epidural Anesthesia given

↳ if Not given then

Mark Neuraxial Anesthesia

Never Give General Anesthesia

ECLAMPSIA

Preeclampsia + Seizures

↳ GTCS (Grand mal)

1st seizure ⇒

Antepartum (M/C)

Intrapartum

Postpartum ⇒ It Means Clin 48hrs
seizure comes

Causes ⇒

Cerebral
hypoxia

+

Cerebral
edema

Rx ⇒

MgSO₄

⇒

Central Action

doesn't do
any peripheral

vasodilation

NMDA
Receptor

→ cerebral vasodilation

* In Periphery $MgSO_4 \rightarrow Ca$ channels

(55)



So; don't club Ca^{2+} -channel blockers (for HIN) & $MgSO_4$



Causes "Neuromuscular Paralysis"

So; $MgSO_4$ gives Σ Labetalol ($anti-HIN$)

*

1st ly - looks for vitals



Stabilize the position (by tie her legs & hands)



Secure Airway — Mouth GAG

Suction

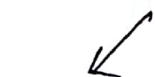
Oxygen by Mask



Inj. $MgSO_4$ + Anti-HIN



Pritchard's Regimen



Loading dose



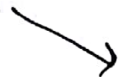
14 gm



4 gm -
12 gm -

Monitor by
Heart Rate

Slow i/v @ 1 gm/min
i/m can be used but not



Maintenance dose



before giving dose



then give 5 gm i/m dose
(@ Alternate site)

given every 4 hourly until 24 hr
after delivery or sub. after Latex allergy test
check \Rightarrow Patellar Reflex \oplus is
RR > 12 /min

Urine output > 30 ml/hr
to check oliguria

If Any of the Patellar Reflex \oplus or
RR $> 12/\text{min}$
Urine output $> 30\text{ml/hr}$ } Any Absent
↓
Omit the dose
↓

Send serum Mg^{+2}

↓
Therapeutic Level = 4-7 meq

(a) 9-12 meq }
(a) 10 meq = Patellar Reflex Absent } warmth
diaphoresis;
(a) 12 meq = Respiratory distress } slurring of
speech

Antidote for $\text{MgSO}_4 \Rightarrow 10\text{ml of } 10\% \text{ 1hr Ca gluconate}$

* Indication of Prophylactic $\text{MgSO}_4 \Rightarrow$ In Impending Eclampsia.

In HELLP syndrome;

In Severe Preeclampsia

* Oliguria is Not a toxicity symptom of MgSO_4 .

Q. Q. Primigravida 34 weeks gestation; Casualty with
headache x 4hrs; 2 episodes of vomiting o/e BP=160/110
mmHg; FHR-(N); Most appropriate step of Mx (56)
Fundal height = 34 weeks; Urine output Protein (2+).

(Firstly) MgSO_4 + Antihypertensive + Top
(\equiv) \rightarrow then \rightarrow then

* Eclampsia is Indication for TOP

\downarrow

vaginal delivery > C.S.
delivered within 24 hours

DIABETES IN PREGNANCY MELLITUS

Over+

Pregestational

Known case of "Diabetes
Mellitus"

Gestational

1st time deranged
sugars are in pregnancy
diagnosed

— Most common

Single Step

↳ both screening & diagnostic

2hr OGTT

— National guideline DIPSI criteria

— done @ 24-28 weeks

— glucose load 75 gm (oral)

— \pm Fasting

after 2 hrs
75 gm oral
glucose

Blood sample

> 140 - GDM

≥ 200 - overt GDM

$120-140$ = Impaired glucose tolerance
of pregnancy

* All @ Women Must undergo & have OGTT

↳ Universal Screening (57)

* Early testing — 1st Antenatal visit

① if she gives H/o baby \in GCA

② if she gives H/o Still birth

③ if she gives H/o Macrosomic baby

④ if she gives H/o Diabetes — 1st degree

⑤ if she is obese

FBS

FBS
or

HbA_{1c}

Fasting $\geq 126 \Rightarrow$ overt diabetes.

* Diabetes \Rightarrow High Risk

For Foetus

↳ ↑ Risk of Gross Congenital Anomaly by
40% bold in compair to @ ♀

↓

dist hyperglycemia (leto-toxic)

↓

Free Radical Injury

* Risk of GCA tes in over
b/c gestational tes after
24-28 weeks

* M/c GCA \Rightarrow CVS > NTD

* Most specific \Rightarrow Caudal Regression Syndrome
(Sacral Agenesis)

* M/c CVS Anomaly in fetus \Rightarrow VSD
of diabetic Mother

* M/c Specific Anomaly in fetus \Rightarrow TGA
of diabetic Mother

* M/c finding in fetus of diabetic \Rightarrow HOCM
Mother

~~11/11/18~~ Anencephaly

Frog eye sign / Mickey Mouse sign on US

Anencephaly — earliest \Rightarrow 10 weeks

— diagnosed \Rightarrow 14 weeks

* Screening / diagnosis \Rightarrow USG

* Recurrence Risk = 4% ; Recurrence Risk = 10%
In Previous 1 anencephaly In Previous 2 anencephaly
baby baby

* Max^m complication seen in female foetus except \Rightarrow

Megalosomia
Respiratory complication } In Male

(58)

* Anencephaly seen in Polyhydramnios \odot

↳ Post-term Labour > Pre-term Labour
(Initiation of Parturition \Rightarrow CRP)
↳ Mainly Face Maldevelopment seen.

* Banana sign }
Lemon sign } seen in spinal
 bilida & Arnold-
 chiani syndrome.

- Test - for Risk of Anomalies

HbA1c \Rightarrow (a) 6.5 \Rightarrow Risk tes by 3%.

(a) 7.5 \Rightarrow Risk tes by 4%.

(a) 8.5 \Rightarrow Risk tes by 8%.

> 9 \Rightarrow Risk tes by 15%.

* USG \Rightarrow for Anomalies diagnosis \rightarrow Fetal imaging for fetal anomalies
↳ (a) 18-20 weeks ↳ Level-II (TIFFA)

* Level-II USG do for all Preg. women

Prevention of Anomaly \rightarrow

[illegible]

High + glucose control

- Give Folic Acid Supplementation

over + diabetic \rightarrow 4mg

previous H/O M/D

Antiepileptics

Prophylaxis of IFA

MMO
0.4 mg

0.5 mg

(RDJ)

- When to Start! \Rightarrow as soon as they present preconceptually

* Minm Requirement :- 1 month before to 3 month after

2. Macrosomia

Hyperglycemia $\xrightarrow[\text{placenta}]{\text{cross}}$ Hyperglycemia in ~~transf~~ foetus

↓

Hyper Gnulinemia \leftarrow Stimulates Fetal Pancreas

L Fat deposition — Shoulders

Abdomen

Pederson's by the fire

Diagnostic for Macrosomia



EBW $\geq 4\text{Kg}$



on US

↳ Single - Fetal growth



Abdominal circumference (Macrosomia IUGR)

* US also used to calculate gestational age

Best In 1st Trimester for gestational age = CROWN-RUMP Length

2nd " = Biparietal diameter

3rd " = Femur Length

Best for Gestational Age = CRL

* Best for gestational Age →

① CRL for 16 weeks

~~② BPD @ 16 weeks~~

③ Femur Length 30 weeks

④ Abdominal circumference 30 weeks

* Macrosomia Patient presents \bar{c} Shoulder dystocia
↓

> 1mm delay in the delivery of
Shoulders after delivery of head



TURTLE SIGN



↳ Sudden pulling back of head towards
Maternal Perineum

* Mx of Shoulder dystocia ⇒

H — Call for Help

E — Empty bladder [episiotomy do

L — 1st — Legs — McRobert's Manoeuvre
↳ Sudden flexion & Abduction of Maternal

P — Suprapubic pressure — thigh on the Maternal Abdomen
Not fundus pressure

E — Enter — Wood's screw Manoeuvre
↳ It ↑ space in A-P diameters

R — Removal of Posterior Arm

R — Roll over on 4 limbs



Gaskin all 4 ways

we injured Lateral
Cutaneous Nerve of thigh



Meralgia Paresthetica

Last Manoeuvre ⇒

Zavanelli Manoeuvre

↳ Push head back & do C.S.
into mother

* Theoretically - destructive procedure - Symphysiotomy
 - Cleidotomy (60)
 - Iatrogenic # clavicle

* M/c fetal injury in shoulder dystocia \Rightarrow Brachial plexus

* M/c Maternal complication in shoulder dystocia \Rightarrow PPH

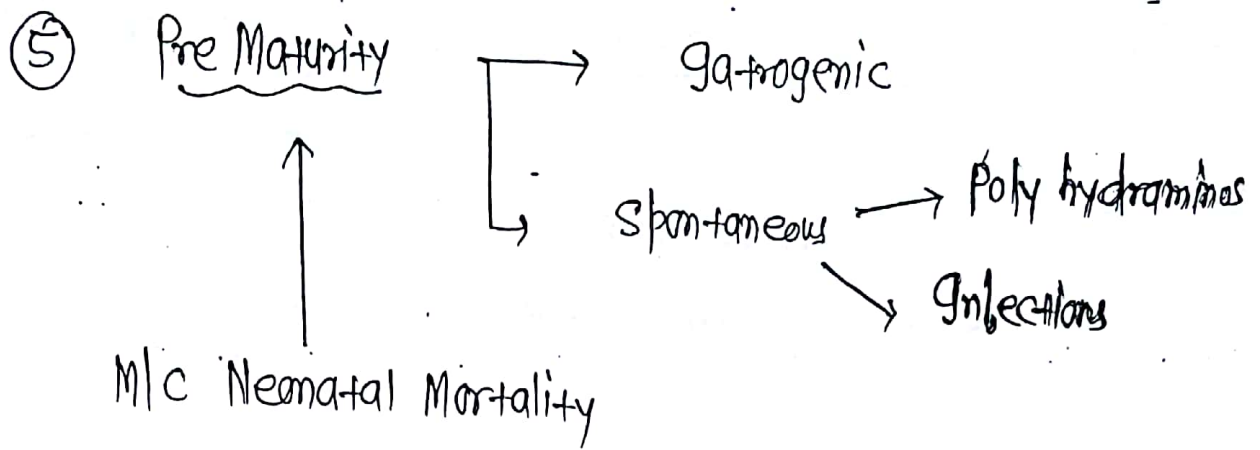
3) GUR \Rightarrow Rare (common)

$\left\{ \begin{array}{l} \hookrightarrow \text{In diabetic vasculopathy} \\ \text{Pre-eclampsia} \end{array} \right\} \text{Utero placental insufficiency}$
 \hookrightarrow Expected birth weight $< 10^{\text{th}}$ Percentile

④ Still birth \Rightarrow Seen in Macrosomic baby, Male baby
 (↑ O₂ Requirement)

\downarrow
 Hypoxic episode

\hookrightarrow Sustained - still birth



⑥ Lung Maturation is delayed

↳ Phosphatidyl glycerol =

Not L/S Ratio

⑦ operative delivery

↳ ↑ Risk of Respiratory Problem

⑧ Immediately after delivery

i) Hypoglycemia

ii) Polycythemia

iii) Hyperbilirubinemia

iv) Hypocalcemia

v) Hypomagnesemia

vi) RDS

vii) HOCM

} Prematurity

* Maternal complication of Diabetic Mother ! →

i) Abortion ⇒ Uncontrolled diabetes

(61)

ii) Polyhydramnios causes → GCM
 → ↑ glucose in Amniotic fluid
 → Hyperglycemia in fetus
 (Polyuria)

iii) oligohydramnios (uncommon)

seen in → Diabetic vasculopathy } in Utero-placental
 Pre-eclampsia } Insufficiency

iv) Pre eclampsia ⇒ 15% Risk Tes

v) Infections — UTI
 — Candidiasis

vi) Complication of diabetes — Vasculopathy
 — Retinopathy
 — Nephropathy
 → Pre existing = worsen in pregnancy

vii) operative deliveries

```

graph TD
    A[operative deliveries] --> B[vaginal]
    A --> C[C.S.]
  
```

viii) PPH (over distension)

ix) Pt. can develop later overt diabetes (in 50% cases)

follow up → 6 week (Post partum visit) | 12wk

by OGTT test

* Management of Diabetes Mellitus in ♀

Maternal Monitoring

- Blood Sugar Level

overt diabetes



Pregnancy



Insulin

GDM



only diabetes diet

40% = Carbohydrate

40% = Fats

20% = Proteins

↓ after 72 hr

do Sugar profile

Fetal Monitoring

- Start to Monitor at 32wk

DFMC (daily fetal Movement Count);

NST

BPP (Biophysical Profile)

USG

; Doppler Never use

only in

GUR

UTI

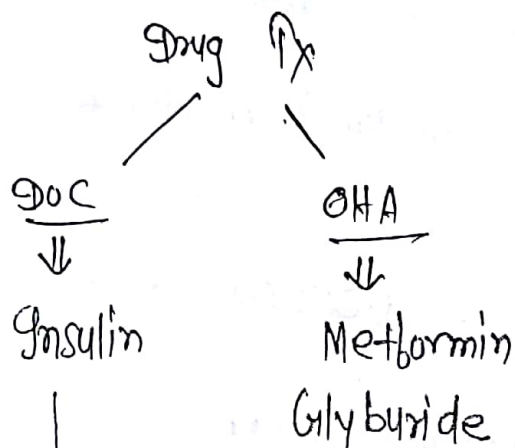
↓
Sugar Profile

Fasting ≥ 95 or

1 hr PP ≥ 140 or

2 hr PP ≥ 120

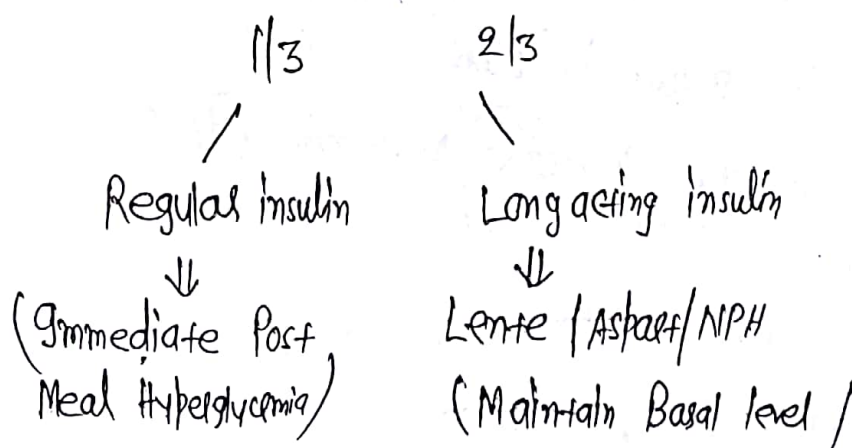
(62)



↓ It doesn't cross placenta.

M/c Insulin use \Rightarrow Regular Insulin

Total dose



1-12 wk = 0.7 U/kg

12-28 wk = 0.8 U/kg

28-34 wk = 0.9 U/kg

≥ 35 wk = 1.0 U/kg

Target Levels

F < 95

1 hr PP < 140

2 hr PP < 120

Avg. glucose ≈ 100

HbA_{1c} $\leq 6.5\%$

* TOP = Gestational diabetes = EDD = 40 weeks
on diet alone

over + GDM on Insulin = 39 weeks

* Diabetes (Alone) is Not an Indication for C.S

* Expected birth weight ≥ 4.5 kg in a diabetic pregnancy
↓

Cesarean section

* In Non diabetic pregnancy ≥ 5 kg

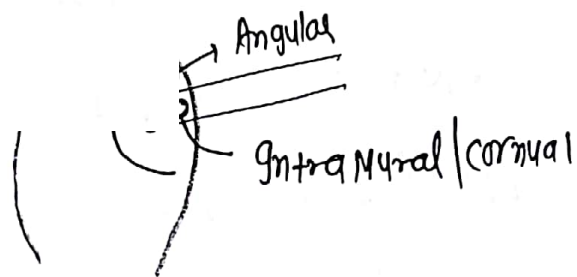
* Insulin Requirement in Labour \rightarrow ↓

↓
Stop Insulin in Labour

& do Intensive Monitoring @ 2hrly.

ECTOPIC PREGNANCY

- Any Pregnancy is implanted outside the uterine cavity. (63)
- Cornual Pregnancy → ectopic ♀
Intramural part of Fallopian tube
- Angular Pregnancy → Intramural ♀
gle of uterus



* Round Ligament is attached lateral to the Growth of Uterus

↳ Angular ♀

* Round Ligament is medial to growth of uterus

↳ Cornual ♀

* Heterotopic ♀ ⇒ 2 Pregnancy simultaneously @ different sites of implantation.

Most common ⇒ Intramural + Intraligament (d/t 3rd sem)

- Risk factors -

- i> Highest Risk of ectopic - H/o Previous Ectopic
 - 1 Previous History $\xrightarrow{\uparrow \text{Risk by}}$ 15%
 - 2 Previous History $\xrightarrow{\text{"}}$ 30%
- ii> 2nd highest Risk - H/o Tubal Surgery
- iii> M/c Risk factor - PID
- iv> Cervicitis
- v> Infertility
- vi> ART (GVF)
- vii> Smoking
- viii> Previous C.S
- ix> Contraception & Ectopic

L All Contraceptive \downarrow Absolute Risk of Ectopic;

certain

\uparrow Relative Risk of Ectopic

\rightarrow Tubal Ligation > IUD > POP

Intrauterine
Device
Progestin
terone
Oral Pill

doesn't inhibit ovulation

so: More Risk of \emptyset (ectopic)

Order of gud in vesing order of Tes Relative Risk
 of ectopic \Rightarrow Progestasert $>$ Mirena $>$ CuT (64)
 \Downarrow

It has gud & Progesterone
 both.

* M/c site of ectopic \Rightarrow Fallopian tube $\begin{cases} \text{M/c site} = \text{Ampulla} \\ \text{L/c site} = \text{Isthmus} \end{cases}$

* L/c site of ectopic \Rightarrow C.S. Scar $>$ Cervical $>$ Abdominal
 \downarrow
 $< 1\%$

* M/c outcome of Ampullary ectopic \Rightarrow Tubal Abortion

2nd M/c outcome of Ampullary ectopic \Rightarrow Rupture @ 8th week

* M/c outcome of Isthmic ectopic \Rightarrow Rupture @ 6th week

* M/c time of Intestinal ectopic \Rightarrow Rupture = 12th week
 or
 Intramural

\Downarrow
 Most Life threatening

\Downarrow
 Hemoperitoneum $\uparrow\uparrow$

* Triad of ectopic \Rightarrow seen in soft patient

Most common
Most consistent

Pain.

Amenorrhoea



bleeding

- H/o Nausea; Vomiting

Shoulder tip pain

↳ Hemoperitoneum

↳ Diaphragm



Referred Pain via Phrenic Nerve

- Syncopal Attack

- Pel Abdomen examination \Rightarrow Tenderness in Lower Abdomen



Rigidity/Guarding

d/t Peritonitis



i.e. Ruptured ectopic

Pel vaginal examination \Rightarrow Uterus - Enlarged; less Period of gestation

Cx Motion tenderness

Salpingitis



differentiate by plan \Leftarrow PID \Rightarrow close d/o for ectopic.

Adnexal tenderness

Adnexal Mass QQ

127
(65)

Investigation \Rightarrow UPT \oplus ve @ 99% times

Investigation of Choice \Rightarrow Trans vaginal ultrasound



1st finding ; which raises suspicion

\hookrightarrow empty uterus

finding ; which raises suspicion against

Not diagnostic
of ectopic

\hookrightarrow complex
Adnexal Mass.

\hookrightarrow Ring of fire sign
on Doppler study

diagnosis finding in ectopic ϕ = Extra uterine gestation
Sac + cardiac activity.

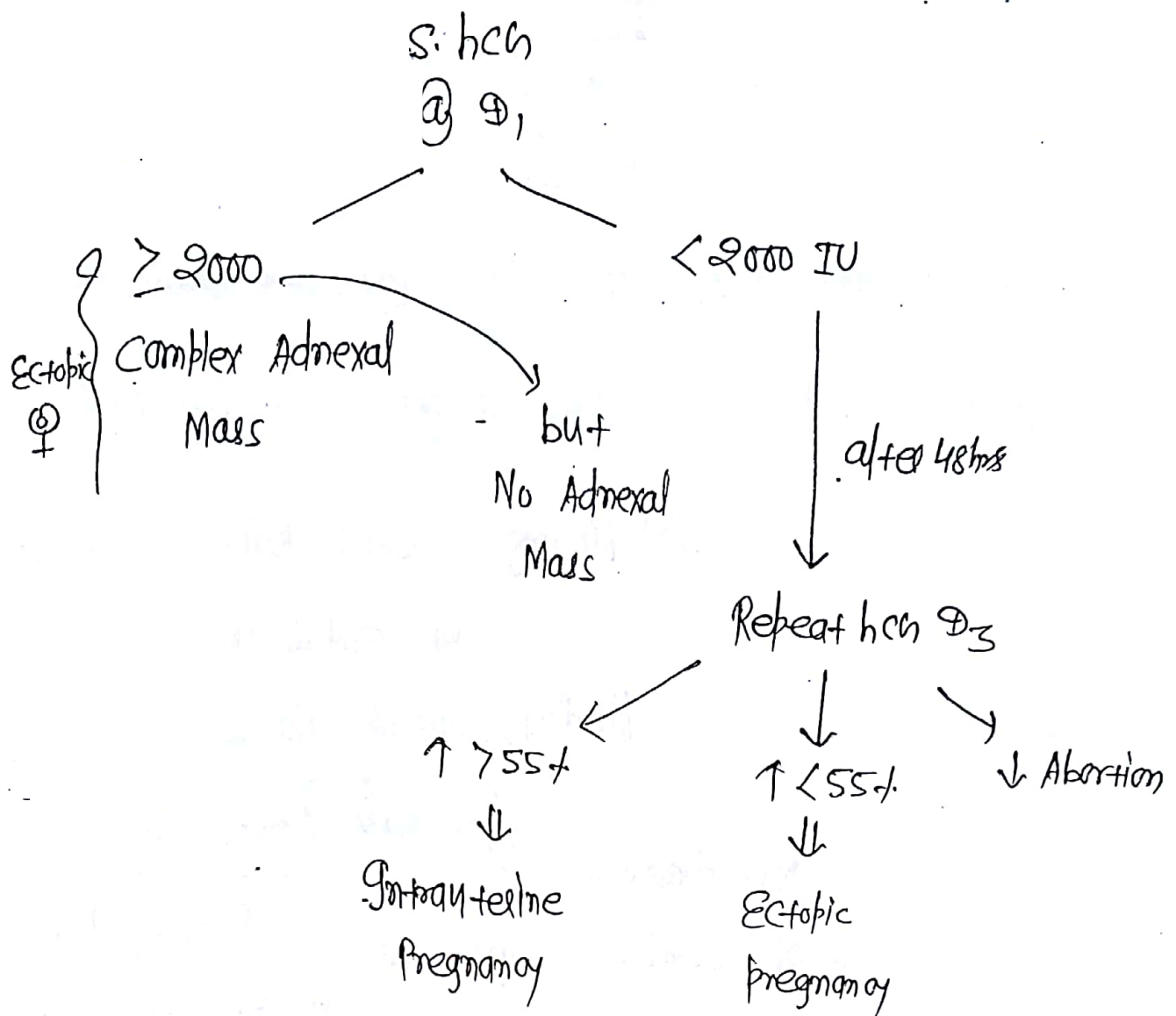
QQ

No Intra Uterine Gestation Sac ; B/L Adnexa (N) ;

Please correlate clinically.



Pregnancy of Unknown origin ; check S. hcg on 9,



* Repeat hcg till it crosses the critical value

* GOC = TVS + Serial β hcg
(=) (=)

* Gold Standard test = Diagnostic Laparoscopy

* Serum Progesterone = 725ng — Live Intrauterine ♀
< 5ng — Abortion

* Culdocentesis
 ↳ if small collection ⇒ USG; if large collection ⇒ Pt. variable
 ↳ Ectopic (ruptured) ⇒ Significant

* Which test has No Role In Ectopic @ \rightarrow

(66)

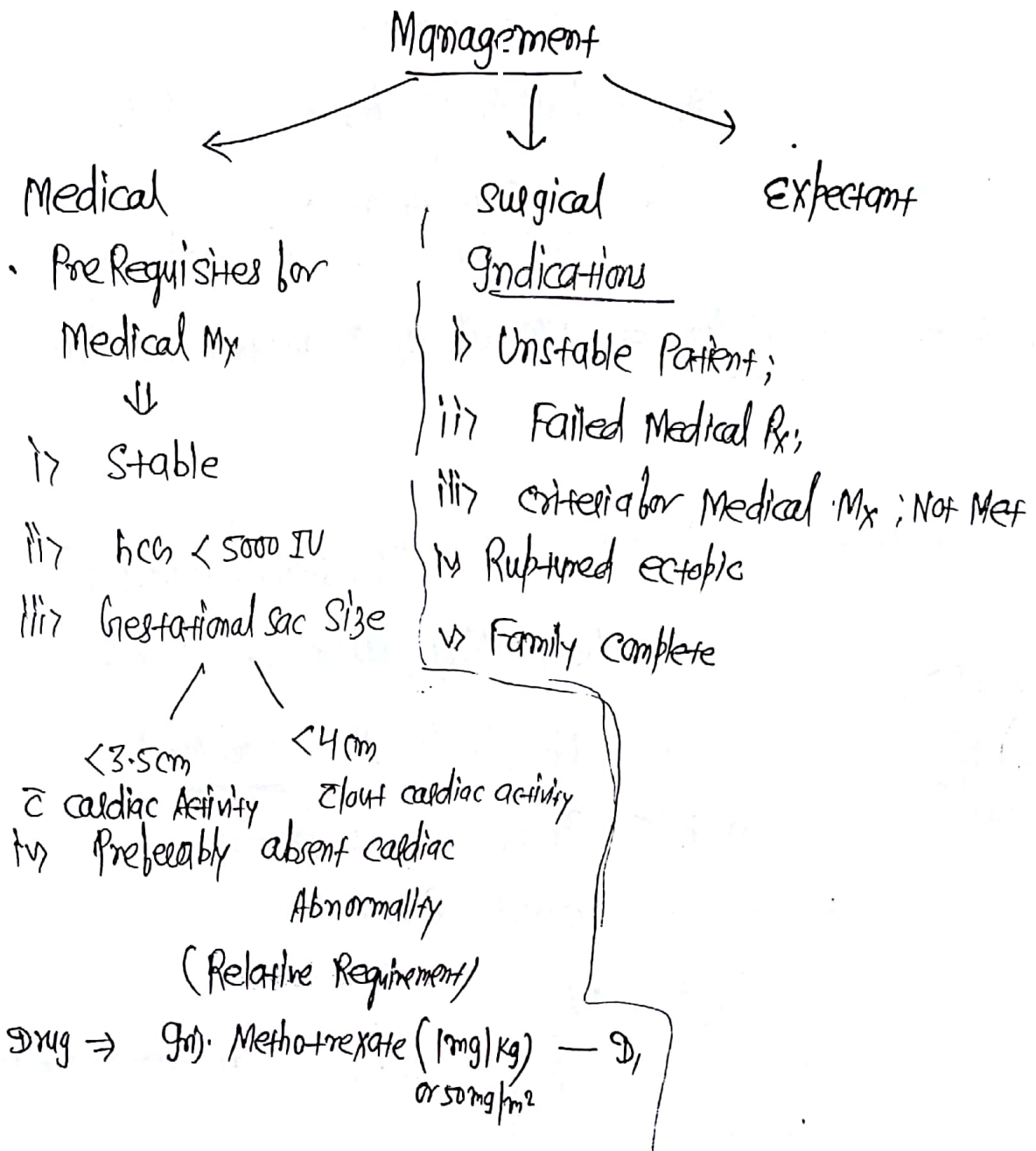
i) Colotomy

ii) HSG

iii) Hysteroscopy

} eliminate patient

is all



D₄ S. hch

Management is Successful if

D₇ S. hch

S. hch level fall by 15% from D₄ value

if D₇ value fall but less than 15% from D₄ value



give Inj. Methotrexate (D₁)



D₄



D₇ (if again less than 15% of D₄ value)

↳ give Inj. Methotrexate

Failed Medical Mx ⇒ Total of 3 Methotrexate Injections

Surgical Mx ⇒

Unstable vitals pt.

Next Most appropriate step ⇒

a) i/v fluids & Medical Mx *

b) ~~if~~ Immediate Lapro-tomy

c) Serial B hch

~~if~~ FAST

if localizing sign ⊕ eg ⇒ peritonitis — do Immediate Lapro-tomy

if No localizing sign ⊖ — do FAST

i) In general Laparoscopy is preferred over Laprotomy. (67)



but if Unstable vitals $\oplus \Rightarrow$ Go for Laprotomy.

ii) Salpingostomy - is preferred over Salpingectomy



Small Incision (1cm)

on Anti-Mesenteric border

(No closure of Incision)

↳ b/c chance of ectopic

↑es by Suturing; suturing heal

by fibrosis & Fibrosis ↑es chance

of ectopic \oplus

i) Family complete

ii) Ruptured ectopic

iii) $\geq 5\text{cm}$

iv) if you can't achieve hemostasis

Q.Q.

Gyn of Marriage — Infertility — Conceived. Rupture ectopic

a) Medical Mx

b) Salpingostomy

~~c) Salpingectomy~~

d) Expectant Mx

Partial



Later date

↳ Tubal Reanastomosis.

* Expectant Management \Rightarrow

Pt. is stable
 β HCG < 200 IU & falling trend
No visible gestational Sac
 \rightarrow Monitor serial β -hcg

* Heterotopic Ectopic \rightarrow

Risk factor = ARM (GvF)

\rightarrow Missed on USG

\downarrow

late diagnosed \Rightarrow by 16 weeks

\rightarrow Big size Sac
 \rightarrow Impending Rupture / Ruptured

TOC: Medical Mx is C/I;

Sx \Rightarrow Laproscopic salpingectomy

If we want to give drug in clinical trials \Rightarrow Inj. KCl

\downarrow
under USG guidance \leftarrow cardiotoxic
give to ectopic child & kill.

* cervical ectopic

(68)

- ↳ Painless bleeding
- ↳ Medical Mx (as long as Pt is stable)
- ↳ Criteria for Dx of cervical ectopic
 - ↳ Paltman's (Rubins criteria)

* Abdominal ~~ectopic~~ ^{ectopic} ⇒ Pain ; No Bleeding

- Studdiford criteria
- Late - 3rd trimester (Abd. ♀)

- 32 week Abd. ♀



Immediate Laprotomy

- ↳ deliver the baby + Placenta in situ
 - ↳ Leads to Autolytic digestion

* Ovarian ectopic ⇒ Speigelberg criteria

- ↳ Surgical Mx

ABORTION **

weight of baby @ 20 weeks
4 300gms

< 20 weeks

≤ 500gms (wkb)

Spontaneous Abortion
(Sporadic)

Recurrent Abortion
(Habitual)

→ means spontaneous

≥ 3 consecutive Preg. losses
< 20wk.

Most common cause of Abortion



Chromosomal

New guidelines

≥ 2 losses documented
↓ confirmed ♂.

Start evaluation

also in 1st & 2nd trimester Most common.

* 50% of total abortion in 1st trimester } Chromosomal
35% of total abortion in 2nd trimester }

* Chromosomal
↓
M/c chromosomal anomalies cause Abortion
Aneuploidy (alteration in No. of chromosome)

M/c Aneuploidies cause Abortion

Trisomy (13, 16, 18, 21)



Single M/c/c

Monosomy X - 20% Abortions

Trisomy 16 - 16% Abortions

QA - M/c cause of Abortion =

a) Trisomy 16

b) Monosomy X

c) Aneuploidy

d) Tetraploidy

* Most viable Aneuploidy = Trisomy 21

Most Lethal Aneuploidy = Trisomy 16 (69)

* M/C caused Recurrent \emptyset Loss \Rightarrow Idiopathic (75% cases)
(RPL)

↓
APLA - 16% RPL

↓
Uterine Anomalies — Congenital
Acquired

↓
Endocrinopathies

M/C \Rightarrow Balanced Robertsonian
Translocations

↓
Chromosomal - only 4% cases of
RPL

* Which of the following doesn't cause RPL?

1) Infections — sporadic

↓
TORCH Infections

Can't cause Early
RPL

Which Infection can cause RPL \Rightarrow "Syphilis"

↓
Mainly cause

1st child $\xrightarrow[\text{Loss } \emptyset]{\text{Pos}}$ 1st child

2nd " $\xrightarrow{\text{"}}$ 2nd child

3rd " $\xrightarrow{\text{"}}$ 3rd child

4th " $\xrightarrow{\text{"}}$ Live born child
(Stigmata syphilis)

Karlson's Law Still birth,
Not Abortion

Every Successive \emptyset in syphilis pos of Loss
Keeps Testing

Q. G5 M4 @ 12 weeks present 2 Missed Abortion.
all her previous early losses; all the following investigation
for her evaluation except \Rightarrow

- ① Karyotyping
- ② LAC
- ③ AEA
- ~~④ VDRL~~

* Karyotyping should be done in patient of RPL

* APLA (Anti Phospholipid Antibody Syndrome) \Rightarrow

\hookrightarrow Single Most common cause of RPL.

Diagnosis of APLA \Rightarrow 1 clinical + 1 Lab criteria

\hookrightarrow Any one of the following

i) ≥ 3 \varnothing losses of < 10 wks

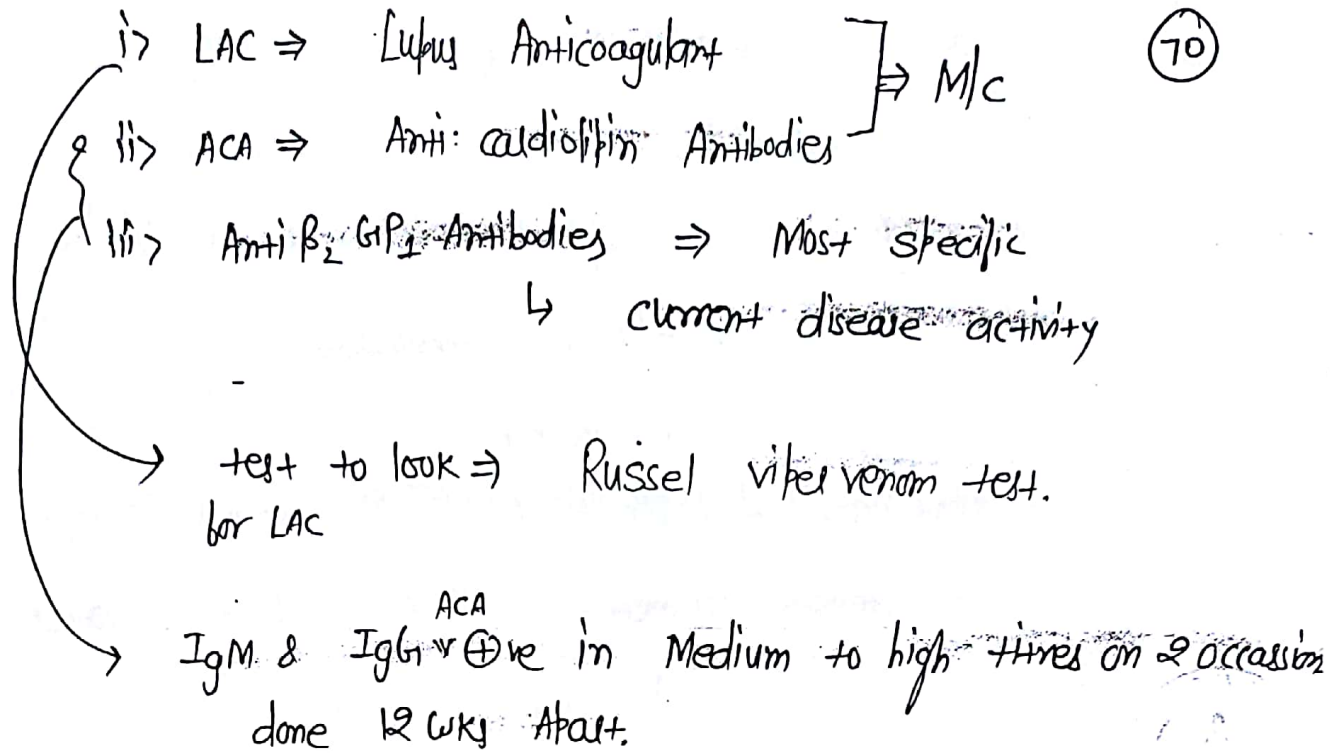
ii) ≥ 1 \varnothing losses > 10 wks of a Morphologically Normal fetus

iii) At least 1 pre-term delivery (< 34 wks)

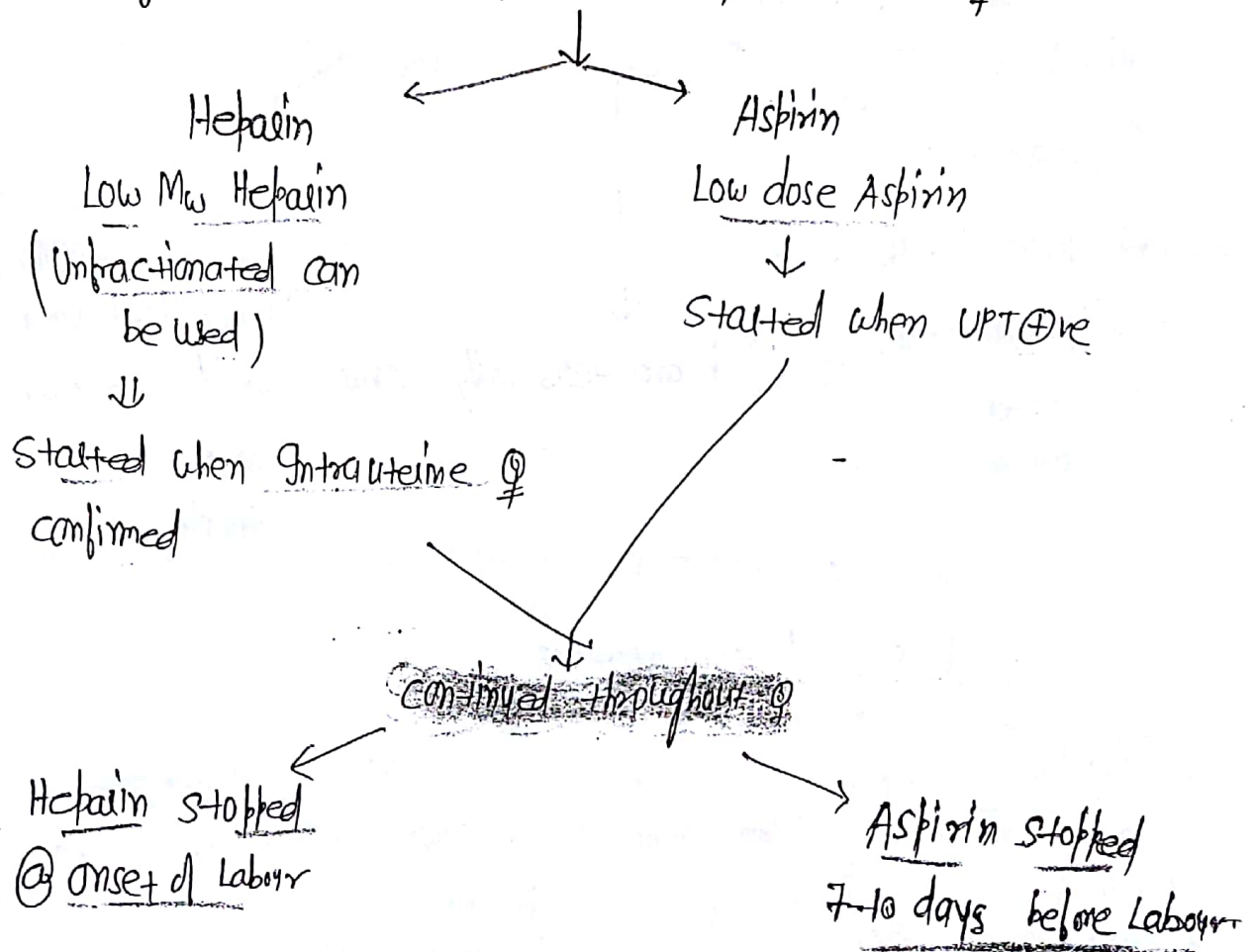
\hookrightarrow Secondary to Severe pre-eclampsia
or Uteroplacental Insufficiency

iv) Venous & arterial thrombosis

Lab criteria \Rightarrow Antibodies



* if the pt. is K/clo APLA syndrome & \oplus also



2nd line \Rightarrow should be used when only 1st line & fails.

\Rightarrow Plasmapheresis

Iv Ig

Doc for APLA \Rightarrow Warfarin (Not given in ϕ)
↳ (cause embolopathy)

Doc for APLA in ϕ \Rightarrow Low Mw Heparin.

* Routine test Antibodies for APLA — In women ϕ RPL

* Most characteristic trimester for APLA — 2nd trimester

* UTERINE STRUCTURAL ANOMALIES CAUSES ABORTION

Congenital

- Septate Uterus

↓

It can even cause
1st trimester abortions

Most characteristically cause 2nd trimester abortion

Acquired

- Cervical Incompetence ^{M/c Acquired cause}

- Fibroid (Submucosal)

- Polyp

- Adhesion

Diagnosis of cervical incompetence

USG based diagnosis

i) do in 2nd trimester & see cervical
Length \Rightarrow if it is $< 25mm$,
& internal diameter $> 2cm$

obvious in subilliac

History based diagnosis

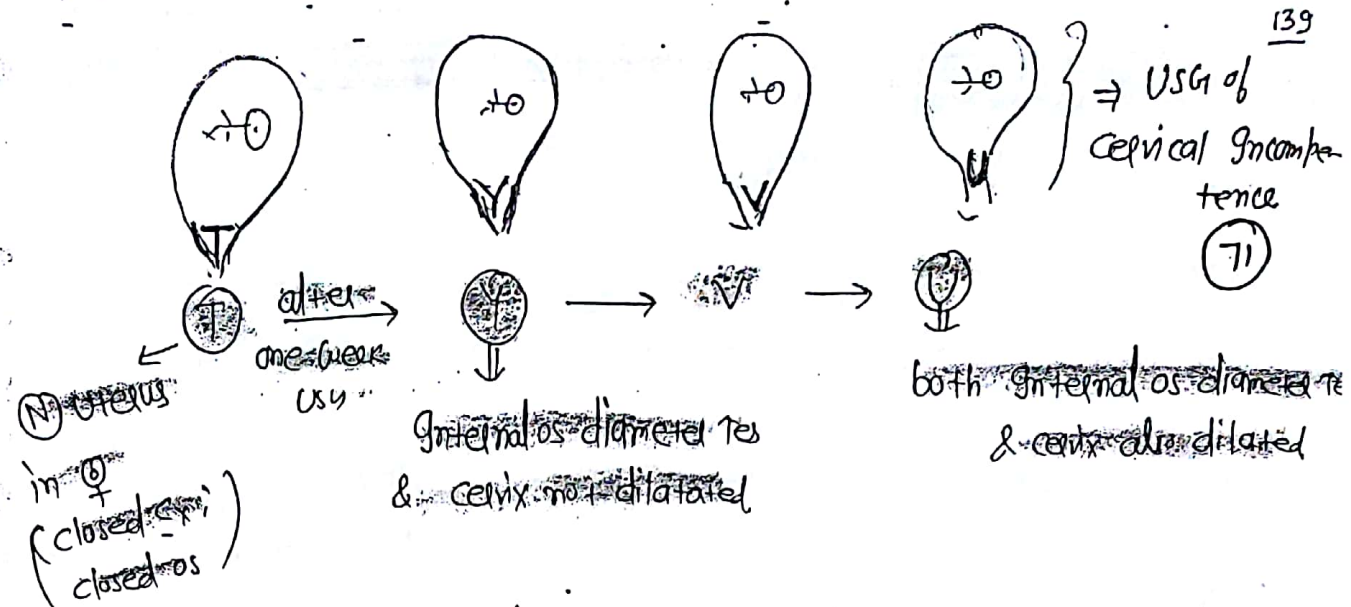
i) cause 2nd trimester abortions

in RPL

iii) Painless dilatation of cervix

↳ in ovar. carcinoma pressure the ph. of less keeps open

(Not cause 1st trimester abortion)



* Diagnosis of cervical Incompetence in Non pregnancy state :

i) Passage of No 8 Haggel's dilator through the Internal os without Resistance

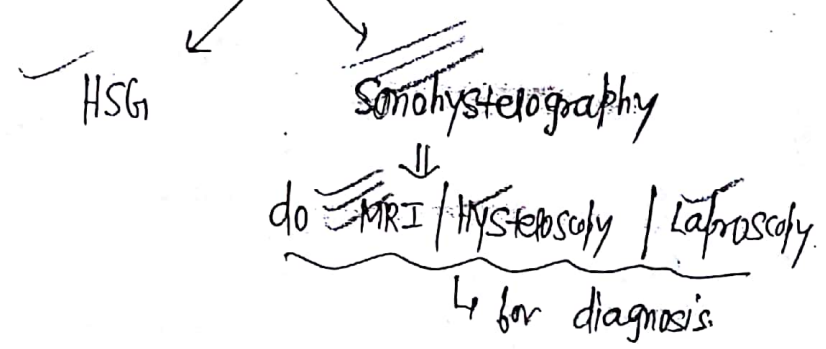
↳ done in premenstrual period

ii) No 16 Foley catheter → fill the balloon with Normal saline

↓
Pull it out without Resistance from the Internal os

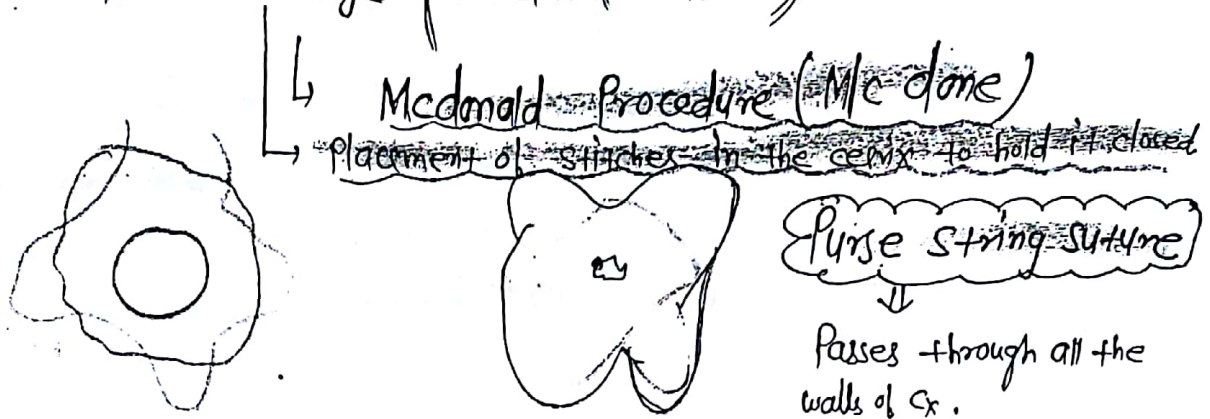
↓
- Cervical Incompetence (+)

* Screening for Uterine Anomalies (Mullerian Anomalies)

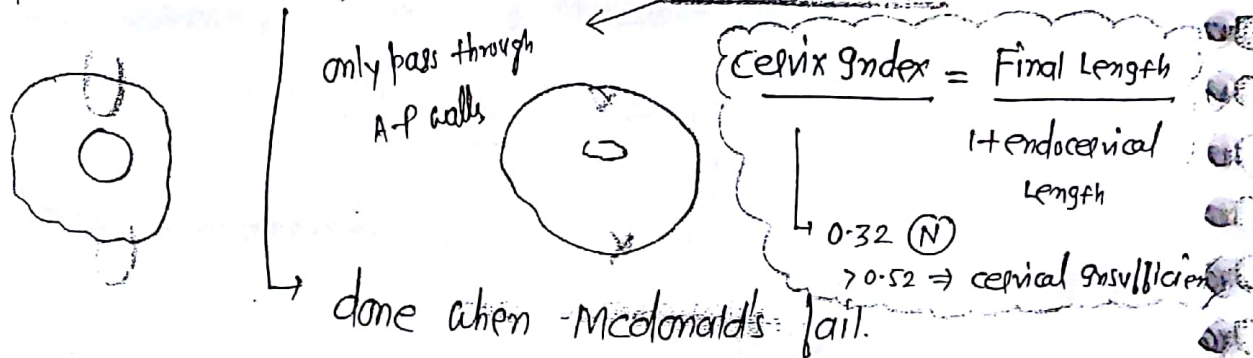


if Pt. has Cx Incompetence ??

do cerclage (cervical stitch)



Shirodkar cerclage \Rightarrow use Merselene tape *



* Ideal time to do \Rightarrow 12-14 weeks (14 weeks)

Up to what time we put \Rightarrow up to 24 weeks
the cerclage

At what level we put \Rightarrow as close to internal os
the cerclage

Contraindication of cerclage \Rightarrow i) GTC

ii) Current pelvic infection

iii) Ruptured Membrane

iv) placenta previa

Removal of Suture \Rightarrow @ 37 weeks

So if any of the following develop \Rightarrow Ruptured Membrane
Chorioamnionitis
Premature labour

* In Non-♀ State ⇒ ~~Lash & Lash Surgery~~
 Rx of cervical Incompetence Lash & Lash Surgery (72)

Qa Should we do Sonohysterography/USG in Recurrent Pregnancy loss

⇓

Yes (~~Should be done as a routine in Non-♀~~)

⇓

In ♀ → do USG

ENDOCRINOPATHIES CAUSES RPL

* Thyroid !! - Hypothyroidism

⇓

do TSH → as Routine Screening for RPL
 ↳ Subclinical Hypothyroidism → Abortions

* Diabetes → Uncontrolled diabetes

↳ Blood sugar is ~~Not~~ a Routine procedure

↳ do when pt. is symptomatic / Significant Family History

* Prolactin → Hyperprolactinemia

S. Prolactin → ~~Not a Routine test~~

M/c presentation in ♀ ⇒ Amenorrhea & Infertility

* L.P.D (Luteal Phase defect) → corpus Luteum is formed by secreting Less Progesterone

↳ Not an established cause of RPL
↳ only when Progesterone < 15 ng/ml

↳ Not a Routine test

* only 4 established cause of RPL

↳ APLA

Uterine Anomalies

Chromosomal

Hypothyroidism

* How does women's Abortion presents ⇒ Spontaneous Abortion

Missed

all presents \bar{c}

Threatened

Amenorrhoea + Pain + Bleeding

Inevitable

* P/S examination

↳ Os closed

as closed

as open

* Size of uterus

POG

POG

POG

* In UGS examination
Cardiac activity is absent
(missing)

Cardiac activity is
present

Q. Lady is 6 week Amenorrhoeic; Pain + Spotting;

USG = Intrauterine gestation Sac & embryo; \bar{c} No cardiac activity.

* if CRL \geq 7mm & absent cardiac activity (73)
 ↳ Missed Abortion. ↳ Non viable pregnancy.

but if CRL 5mm & absent cardiac activity

↳ Repeat USG after some times (Min^m 1 week)

Non-viable @ \Rightarrow



Mean Sac diameter

≥ 25 mm & No yolk/embryo inside

Blighted ovum (No yolk inside b/c of Avascular villi)
 (empty sac)

Q. Missed Abortion is missed b/c Symptoms presented after later date of Abortion; so; we missed to diagnosis.

Mx \Rightarrow

Induce Abortion (to open the os)

↳ In Missed Abortion

9. Empirical Rx (Bed Rest; Progesterone Supplement, i/m - weekly injections, Micronised oral tab)

avoid Intercourse

avoid Lifting heavy weight

\Rightarrow In Threatened abortion \Rightarrow abort d/t chromosomal abnormality

No Specific Rx \Rightarrow In Inevitable Abortion.

** Complete Abortion

Amenorrhoea + Pain + Bleeding + H/o expulsion of fetus
P/S examination

L os closed

↓

Symptoms improve
after expulsion

USG

↓

For confirmation

↓

Uterine cavity empty
No Retained Product of Conception
(RPOC)

** Incomplete Abortion

os open

↓ Period of conception
(Poc's) can be seen in the
cervical canal

Uterine size 2 pots

Bleed a lot

↓ Unstable vitals

(Patient may present c/

Shock)

Mx

Symptomatic Rx

Mx

Specific

complete the process

↓

do Suction & evacuation
(digital evacuation could be
life saving)

* Induced Abortion ⇒ MTP Act

↓

Up to 20 weeks

Pt. Asked for Abortion for ⇒ Rape ⇒ Humanitarian

Contraceptive Failure ⇒ Social

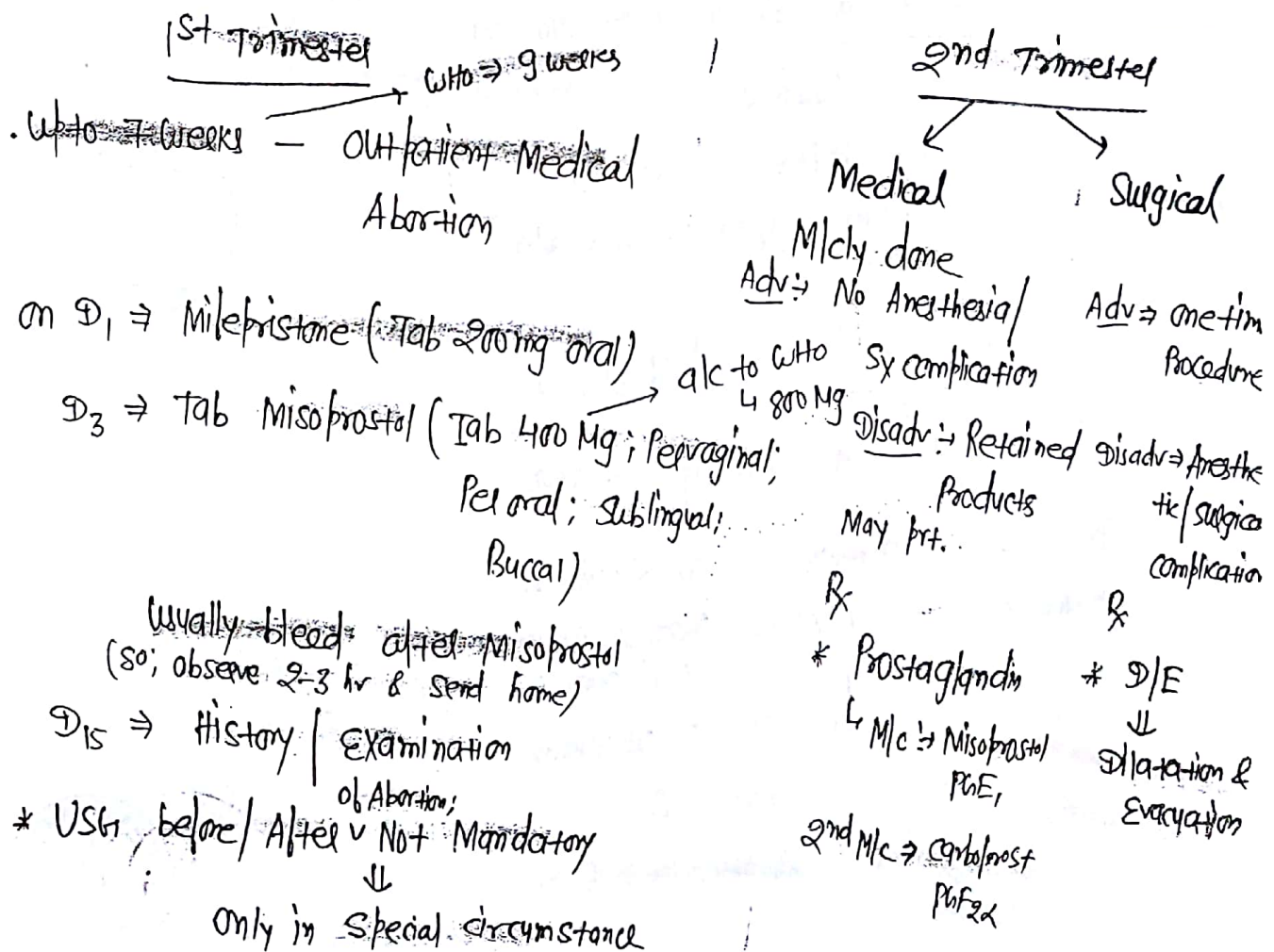
~~Medical~~ → Eugenic
~~surgical~~ → Therapeutic
 disease

(74)

Who do Abortions ⇒ RMP's (Registered Medical Practitioners)
 ↓
 i) degree/diploma in field of OBG;
 ii) 6 months of ~~lowe~~ ^{low} surgical in department of OBG;
 iii) assisted in 25 procedure of abortion in govt hospital (out of 5 done independently)

< 12 week ⇒ 1 RMP

12-28 week ⇒ 2 RMP



• Beyond 7 weeks & upto 12 weeks

↳ do "Suction & evacuation"

↓
• Hegar's dilator ⇒ graduated

blunt devices (if we perforate)

it heals itself; wait & watch

• Karman's cannula ⇒ 600 mm of Hg pressure

↳ white plastic device

generated

Monitor the vitals

↳ on p/v examination

↳ if we perforate by Karman's

cannula ⇒ Immediate lapro-

↳ size of cannula = size of uterus

scopy

on p/v examination to check

Avascular

Necrosis of

bowel loop

↳ usually 1 less than foli's

• End point of Suction & evacuation

→ i) ↓ bleeding

ii) Air bubble in cannula

iii) Gripping sensation on cannula

• Check curettage ⇒ Sharp devices

↳ give grating sensations

In village; alternative to Suction & evacuation

⇒ MVA ⇒ Manual Vacuum Aspiration

←
cannula
bore of syringe (needle)

↳ up to 12 weeks

Syringe = 60 cc

Pressure 600 mm Hg - 600 mm Hg

Medical Rx

Extracannulaic

Ethacridine

Intracannulaic hyperosmotic saline

Oxytocin

Dilatation & Evacuation

147

75

↳ ~~Over~~ Forceps \Rightarrow Spoon shaped forceps
No Locks \oplus
Pieced the products

* MOBIUS SYNDROME \Rightarrow ~~diff. Misoprostol~~

↳ Palalysis of Facial Muscle
(Facial Nerve affected)

* if Cx is Not open after giving Misoprostol & all drugs in
Under 20 weeks

\Downarrow

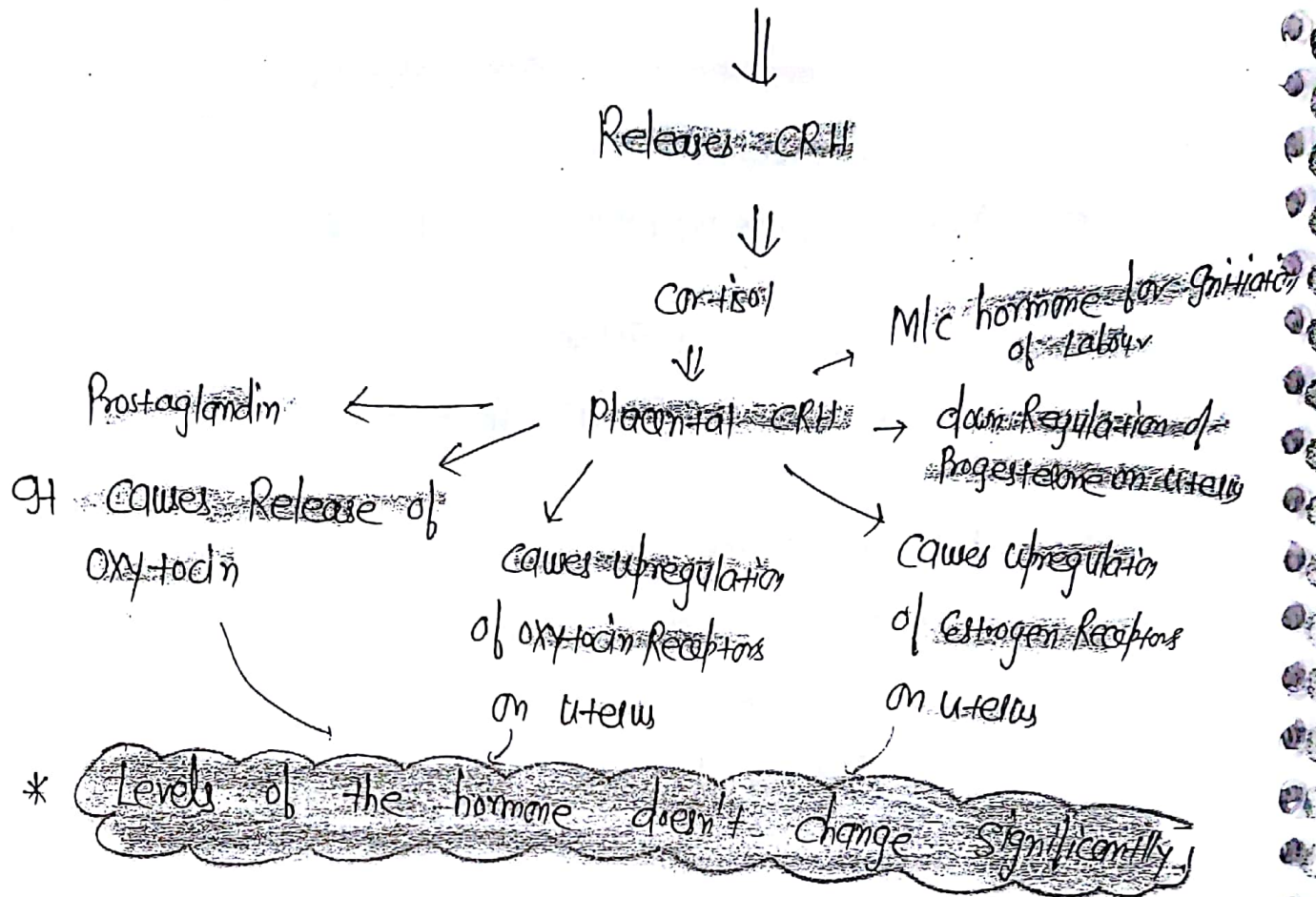
Opening of uterus — In early Gestation

\Downarrow

Hysterotomy
(So; it gives Classical Scar; so; sign
after it always do cesarian
section in Next \odot)
blc @ 20 weeks
we don't dilate
are the Lower
uterine
sign

LABOUR

Initiation of Labour \Rightarrow ~~Functional-fetus Hypothalamic-Pituitary Axis~~



Oxytocin works via \Rightarrow Ca^{2+} influx

PhF₂ from the decaying

PhF₂ from the decaying

Max^m oxy. in reaction ⊕ a) 2nd stage of labour

Expected date of delivery = 40 weeks (Naeglell's formula)

L on EDD \Rightarrow 4th delivery occur

FDD + 1 week \Rightarrow 50% delivery occur

EDD + 2 week \Rightarrow 80% delivery occur

* Preterm Labour \Rightarrow before ~~37 weeks~~

(76)

Term \Rightarrow ~~Early term~~ \Rightarrow 37-38⁶

Term \Rightarrow 39-40⁶

~~Late term~~ \Rightarrow 41-42⁶

~~Post term~~ \Rightarrow ~~43 weeks~~

* Non-Medically Indicated C.S. \Rightarrow don't do before ~~39 weeks~~

* Graevida \Rightarrow No. of times the women has been \otimes .

Parity \Rightarrow beyond period of viability.

Abortion (< 20 weeks)

* G - T - P - A - L \rightarrow currently living children.

Graevida

term (≥ 37 weeks)

Pre-term (20-36 wk)

Q. In twin \otimes None of the Parameters change; except \Downarrow Currently living children

Q. 2nd Pregnancy; 1st was twins 34 wk delivery; Both alive

G₂ T₀ P₁ A₀ L₂

* Patient comes c Uterine contraction

check

False Labour

True Labour

- Uterine contraction
⇒ Not ↑ in frequency
Intensity
Duration

- No Passage of blood mixed cervical Mucus
- No Progressive ex dilatation & effacement

- Uterine contraction ⇒ ↑ in frequency
Intensity
Duration

- It Shows ⇒ Passage of blood mixed cervical Mucus

- Progressive ex dilatation & effacement
↓
in-1.
↳ 9 cm
↳ Fully dilated
↳ 10 cm

In Primigravida → effacement → dilatation

In Multigravida → both at simultaneously

- No Rupture of Membrane
- Sedatives ⇒ Uterine contraction will subside

- Rupture of Membrane
- Sedatives → Pain perception ver. Labour will progress

Best sign ⇒

Progressive ex dilatation

Rupture of Membrane

↳ ble of PROM ⇒ Rupture of Membrane before onset of Labour

~~Box 4 to know~~ PROM \Rightarrow Pel. speculum examination ⁽⁵⁾



- Leaking of Fluid from the OS. ⁽⁷⁷⁾
- pH \Rightarrow Nitrazine paper test
- USG \Rightarrow oligohydramnios
- dye test.
- FFN (fetal fibronectin)
 \hookrightarrow Marker of pre-term labour also

PPROM \Rightarrow Rupture of Membrane before 37 weeks

\Downarrow
Preterm Pre Mature
Rupture of Membrane

Mx \Rightarrow PROM

\Downarrow
> 34 weeks

Induce Labour

(Risk factor of chorioamnionitis)

+

Prophylactic Antibiotics

\swarrow
< 34 weeks

Antibiotics
steroids

\Downarrow

once > 34 weeks

\Downarrow

Induce Labour

Threatened Preterm Lab

\swarrow
< 34 weeks

Steroids + Tocolytics

\Downarrow

No Role of
Antibiotics

\searrow
> 34 weeks

\Downarrow

Wait & Watch

* Most Imp. R/F for Pre-term Labour

\hookrightarrow Previous H/o of Pre-term Labour

\swarrow
Infection

Prophylaxis in high Risk previous pre-term Labour Patients

↳ Progestelone

* if patient is in True Labour, we know about :-

1st Stage

Onset of True Labour Pain
to full dilatation of cervix

2nd Stage

From full dilatation to
expulsion of fetus

Latent
Phase

Active Phase

0-5cm cervical
 dilatation
 (Active)

$\geq 6\text{cm}$

0-3cm (Latent)

$\geq 4\text{cm}$

↓
duration of
Latent Phase

	Normal	Prolonged
Primi	24 hrs	20 hrs
Mult+	8 hr	14 hr

↓
Rate of dilatation
Rate of descent of head

in Primigravida (1.2cm/hr)

in Multigravida (1.5cm/hr)

(N) Progress = 1cm/hr

Primigravida

1cm/hr

Multigravida

2cm/hr

3rd Stage of Labour →

From the birth of child to complete expulsion of placenta

4th Stage of Labour →

Duration of observation of Mother : 1hr

PARTOGRAM

153 B,B,B ⇒ Blood stained
M,M ⇒ Meconium stained
C,C,C,C,C

Top Most part ⇒ tells about fetus (clear Amniotic fluid,

Middle part ⇒ Progress of Labour (Cx dilatation) (78)

Lower part ⇒ tell about Mother

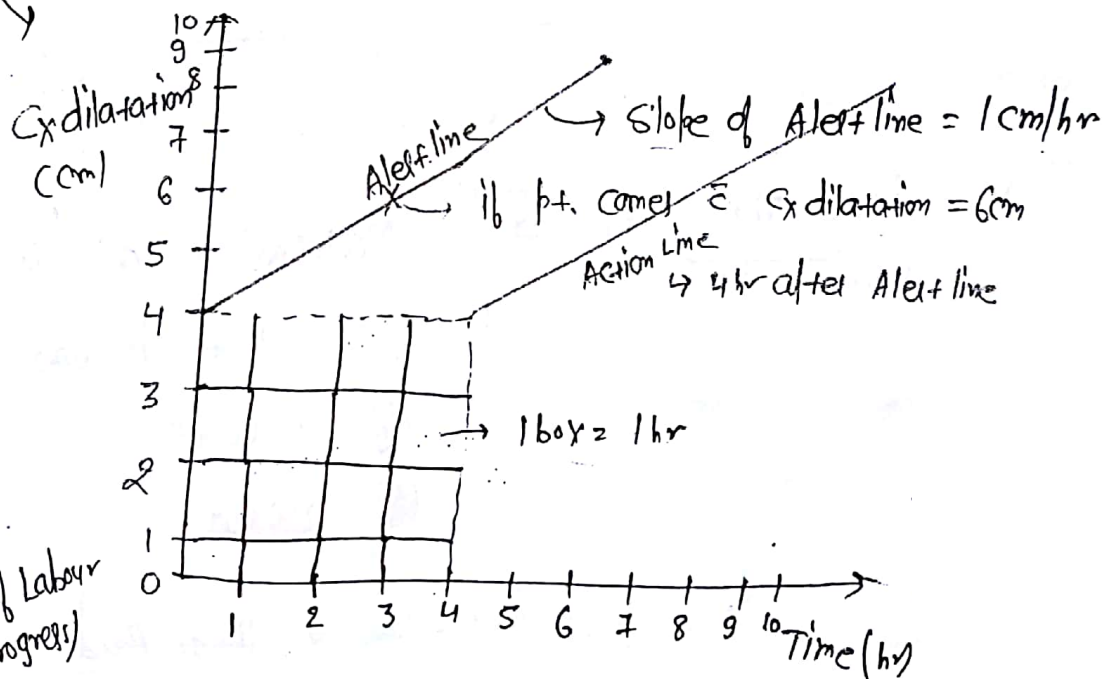
tells about Moulding

Grade 1 ⇒ Parietal bones touch each other

Grade 2 ⇒ overlap; can be separated

Grade 3 ⇒ overlap; can't be separated

Plotting always done on Alert Line



Gynaecia of Labour (Alert Progress)

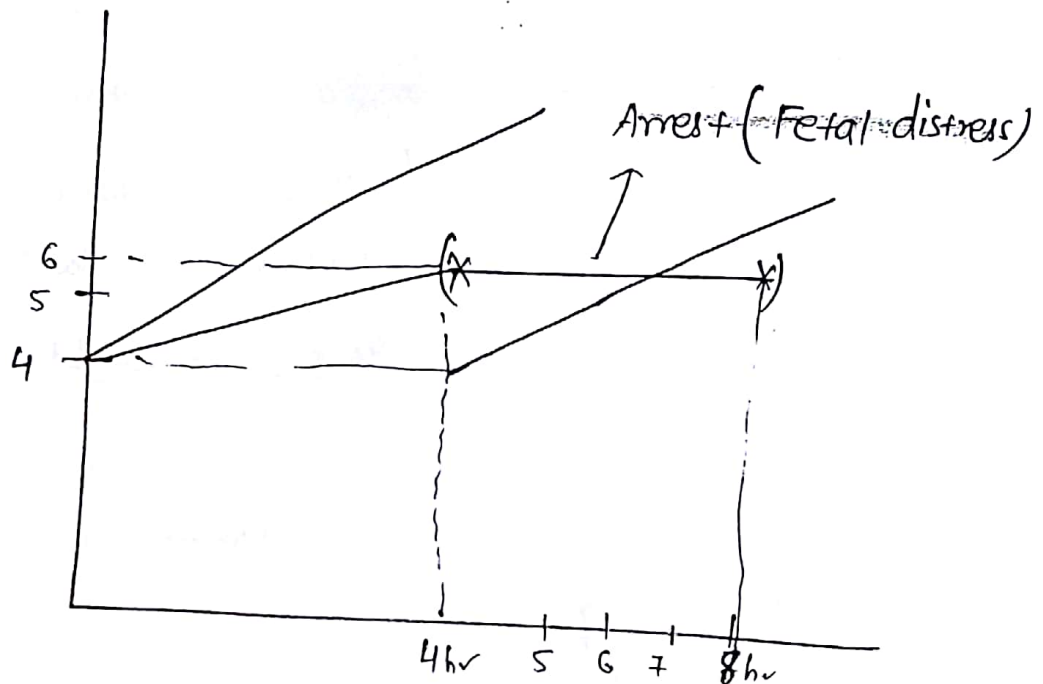
Not plot Latent phase in Partogram (Plot after ≥ 4cm)

Partogram is Right to Alert Line ⇒ Referral to higher center


Partogram goes to Right to Action Line ⇒ Intervention

Arrest of Active Phase \Rightarrow No change in Cx dilatation even after 4 hrs of Adequate uterine contraction \rightarrow only if FHR \Rightarrow (N)


Intervention. Caesarean Section



* Lower Most Part \Rightarrow Maternal condⁿ (uterine contraction)

 < 20 sec in one contraction

 20-40 sec

 > 40 sec

\rightarrow also tells about \Rightarrow Heart Rate
BP
temp
Urine output
drugs to be given

2nd stage of Labour \Rightarrow Avg. duration
 \hookrightarrow 1 hr = Primigravida
 30 min = Multigravida

Arrest of 2nd stage

\hookrightarrow No descent of head in the presence of Adequate uterine contraction

for 3 hrs \rightarrow Primigravida

2 hrs \rightarrow Multigravida

do G.S.

• Arrest of 2nd stage \bar{c} epidural Analgesia

4 hr \Rightarrow Primigravida

3 hr \Rightarrow Multigravida

* Progress of Labour \rightarrow P/A examination \rightarrow Contractions $\begin{cases} \text{duration} \\ \text{frequency} \end{cases}$

\rightarrow descent of head

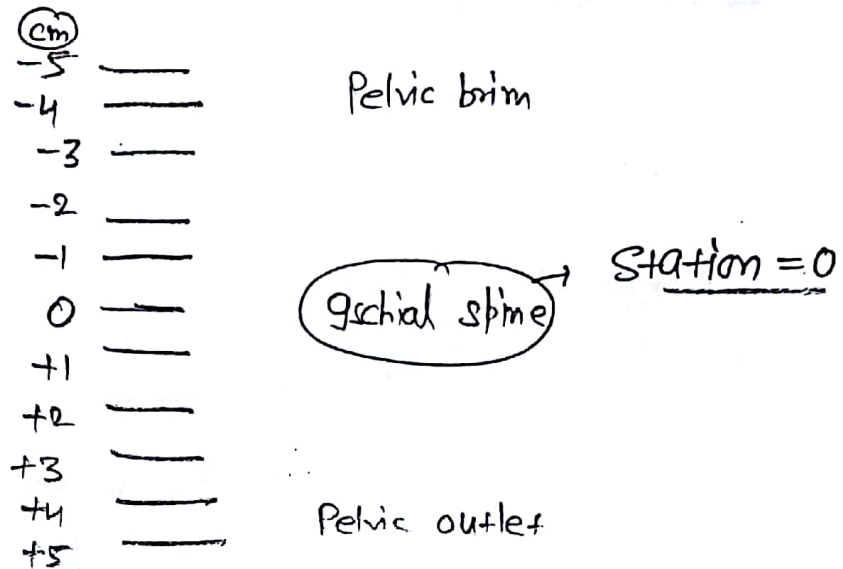
\rightarrow Ruled Five

Numerator	5/5	A \Rightarrow	entire head above pelvic brim
\downarrow	4/5	B \Rightarrow	Sinciput high; occiput easily felt
Parts Palpable	3/5	C \Rightarrow	Sinciput easily felt; occiput felt
	2/5	D \Rightarrow	Sinciput felt; occiput just felt
	1/5	E \Rightarrow	Sinciput felt; occiput not felt
	0/5	F \Rightarrow	None of head palpable

P/V examination \rightarrow Dilatation

\rightarrow Descent of head

Descent of head in p/v examination \Rightarrow



Station -2 \Rightarrow baby's head 2 cm above ischial spine

Station +3 \Rightarrow baby's head 3 cm below ischial spine

* LIE \Rightarrow Relationship of Fetus & Long axis of uterus



firstly correct the detorsion & empty the bladder



Longitudinal



Longitudinal



Transverse



Oblique lie =

Unstable lie



Keeps changing even after 37 weeks

M/cly fundal location of placenta \Rightarrow Conf.

M/c/c \Rightarrow Cephalic ($>50\%$)

2nd M/c \Rightarrow Placenta previa

Polyhydramnios

Oligohydramnios New cause unstable lie

Presentation ⇒

Part of fetus which is foremost in the birth canal.

(80)



Breech



Cephalic



Shoulder

Leopold Maneuvre ⇒

~~Soft, broad part~~ ⇒ ~~buttock~~

~~Smooth curve~~ ⇒ ~~Back~~

~~Limbs~~ ⇒ ~~knobby feel~~

(A) ~~Fundal grip~~ (1st Leopold)

(B) ~~Lateral grip~~ (2nd Leopold) → is with single hand ~~used~~

(C) ~~Pauline's grip~~ (3rd Leopold) → ~~2nd pelvic grip~~

(D) ~~Pelvic/oes/pelvic~~ (4th Leopold) → ~~1st pelvic grip~~

↘ Both hand we use

* If head is in complete flexion ⇒ ~~Vertex presentation~~

Partial flexion ⇒ ~~Shinut presentation~~

Partial extension ⇒ ~~Brow presentation~~

Complete extension ⇒ ~~Face presentation~~

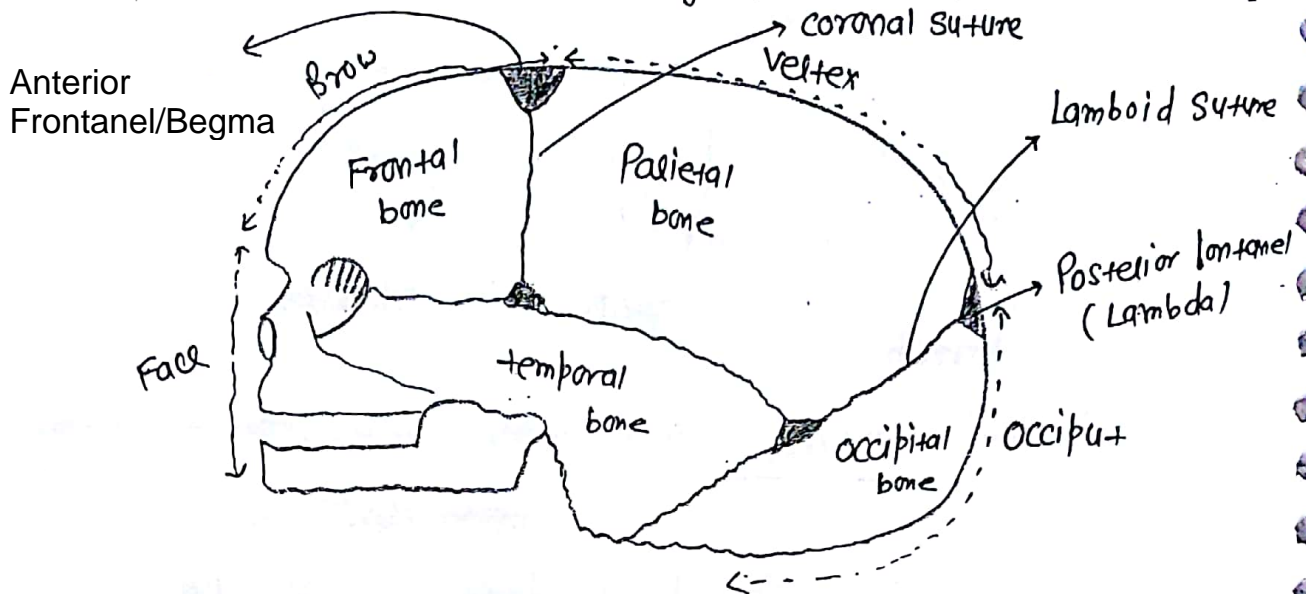
* ~~Anterior fontanel~~ ⇒ ~~Bregma~~

~~Posterior fontanel~~ ⇒ ~~Lambda~~

~~Posterior boundary of Brow~~ ⇒ ~~Anterior boundary of vertex~~

* Anterior fontanel \Rightarrow diamond in shape

Posterior fontanel \Rightarrow triangular in shape



Engagement \Rightarrow When the Largest diameter of the presentation crosses the pelvic brim.

Largest Transverse diameter \Rightarrow Biparietal diameter
 9.5 cm

engaging dm in different presentation \Rightarrow

	Vertex	Sinciput (Partial flexion/Vertex)	complete extension/ Face	Brow
Engaging diameter (AP)	Suboccipito bregmatic (9.5 cm)	Occipito frontal (occiput to anterior end of Anterior fontanel)	Submento- bregmatic (9.5 cm)	Mentoverti- cal (14 cm)
Antelo- Posterior		11.5 cm		Largest dm of fetal head
		Sub-occipito frontal (10.5 cm)		

* In Most of the Primigravida

↳ engagement @ 37 weeks

* free floating head @ 37 weeks in Primigravida

↳ d/t deflected head:

CPD;

Placenta previa;

Polyhydramnios.

* Engagement Rule out cephalopelvic disproportion at the inlet. (CPD)

* when the head is Engaged; station = 0.

↳ on P/A exam; 2/5 is palpable

* DENOMINATOR - Bony point on the presentation used to describe the position of head

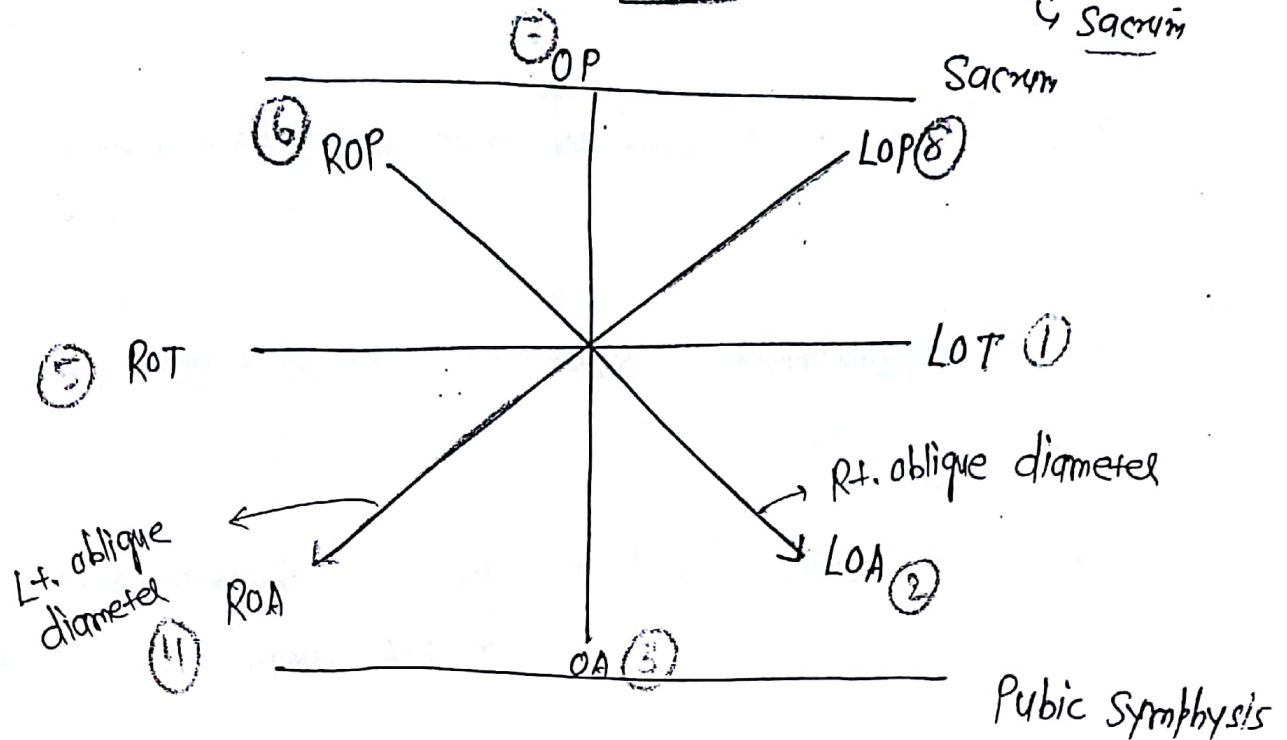
<u>Presentation</u>		<u>Denominator</u>
<u>Vertex</u>	→	<u>Occiput</u>
<u>Breech</u>	→	<u>Sacrum</u>
<u>Brow</u>	→	<u>Frontal bone</u>
<u>Face</u>	→	<u>Mentum</u>
<u>Shoulder</u>	→	<u>Acromion process (Scapula)</u>



Maternal Pelvis →

Occiput ⇒ for vertex presentation

if breech prnt. ⇒ write "s" in place of "o"
 ↳ Sacrum

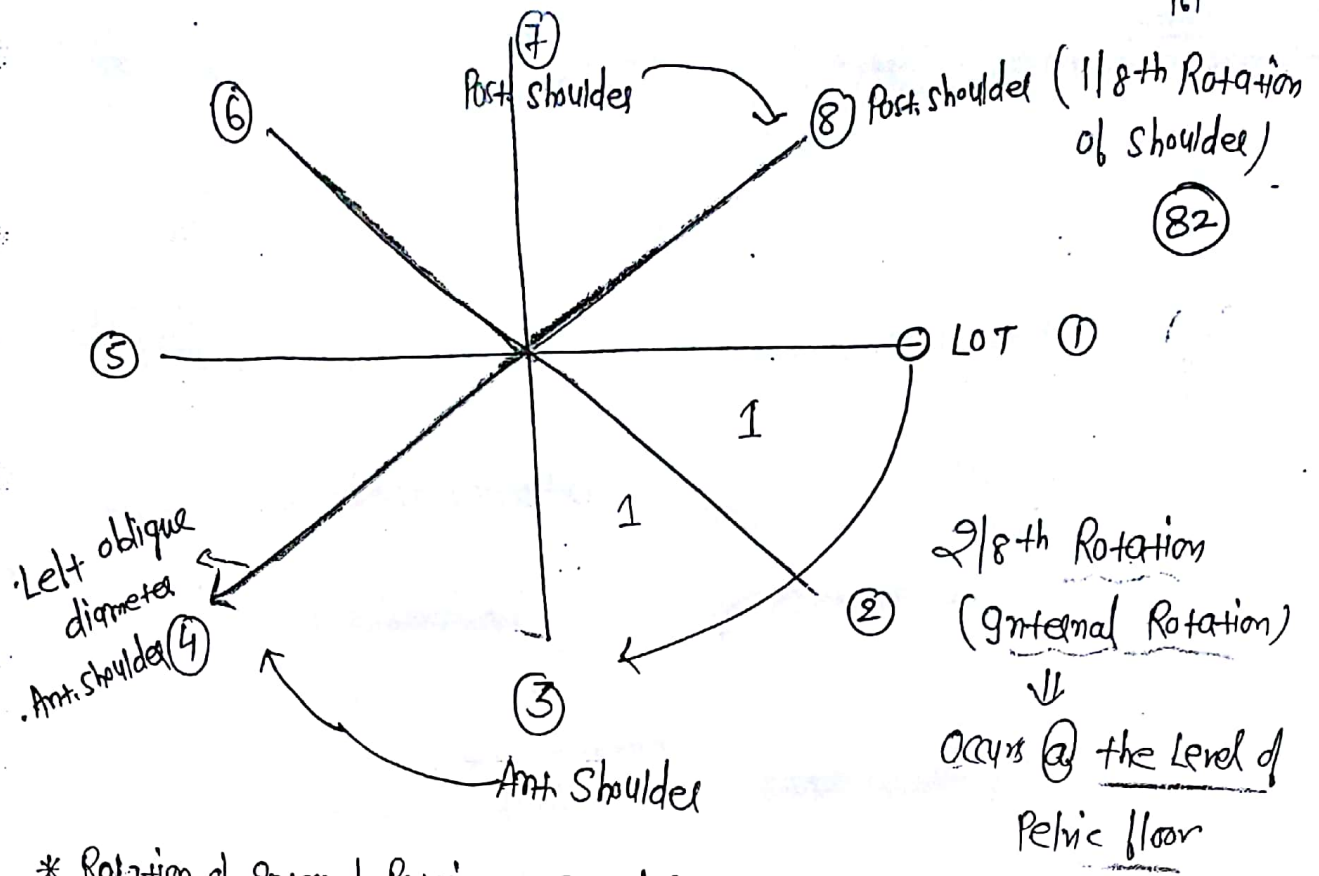


* In Routine exam; Plv exam done in active Labour 4 hourly.

* M/c position of (N) Labour ⇒ LOT**

cardinal steps of (N) Labour

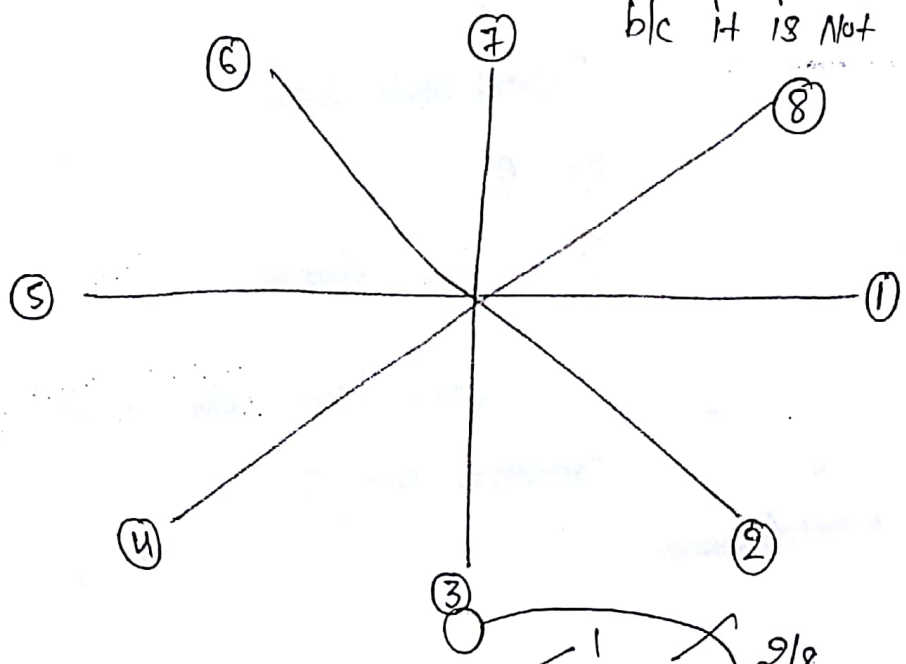
- (1) Engagement
- (2) descent
- (3) flexion
- (4) Internal Rotation
- (5) Extension ⇒ delivery of head (occiput → vertex → forehead)
 (chin → Mouth → Gubella)
- (6) ~~external~~ rotation



- * Relation of Internal Rotation to External Rotation
↳ opposite direction.
- * Relation of Restitution to External Rotation
↳ same direction

but shoulder rotate only by 1/8

* External Rotation : Restitution is Not a cardinal Movement b/c it is Not the separate Movement



after external Rotation head goes back to original position (LOT)

Restitution (to Remove torsion) the head & shoulder

Internal Rotation of shoulder

3 "P"s" Require for (1) labour \Rightarrow

Passage

Push

Passenger

Passage \Rightarrow

Maternal Pelvis \Rightarrow by cardinal planes

M/C pelvis in ♀ = Gynaecoid pelvis (circular pelvis)
 \downarrow
 on the basis of inlet
 4 in 50 ♀



Transverse diameter \geq AP diameter
 = Anthropoid pelvis



Antero-posteriorly oval pelvis
 in 25% ♀

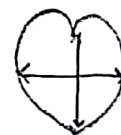


AP diameter
 \downarrow
 Transverse diameter

Other = Android pelvis



Typical Male pelvis
 20% ♀



Transverse diameter
 \downarrow
 AP diameter

Inlet = Heart shaped

Least common pelvis in ♀ = 5+ Coxa (platypelloid pelvis)



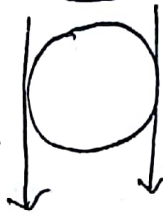
Flat Gynaecoid

Transversely oval inlet



Transverse diameter \gggg AP diameter

Gynecoid



Side wall - parallel

Ischial spine \Rightarrow Blunt

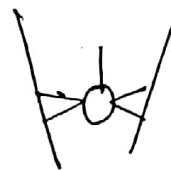
• DTA Not Seen

Subpubic Angle \Rightarrow obtuse
(90-100°)Shallow Pelvis

Android (Male Pelvis)



convergent side wall

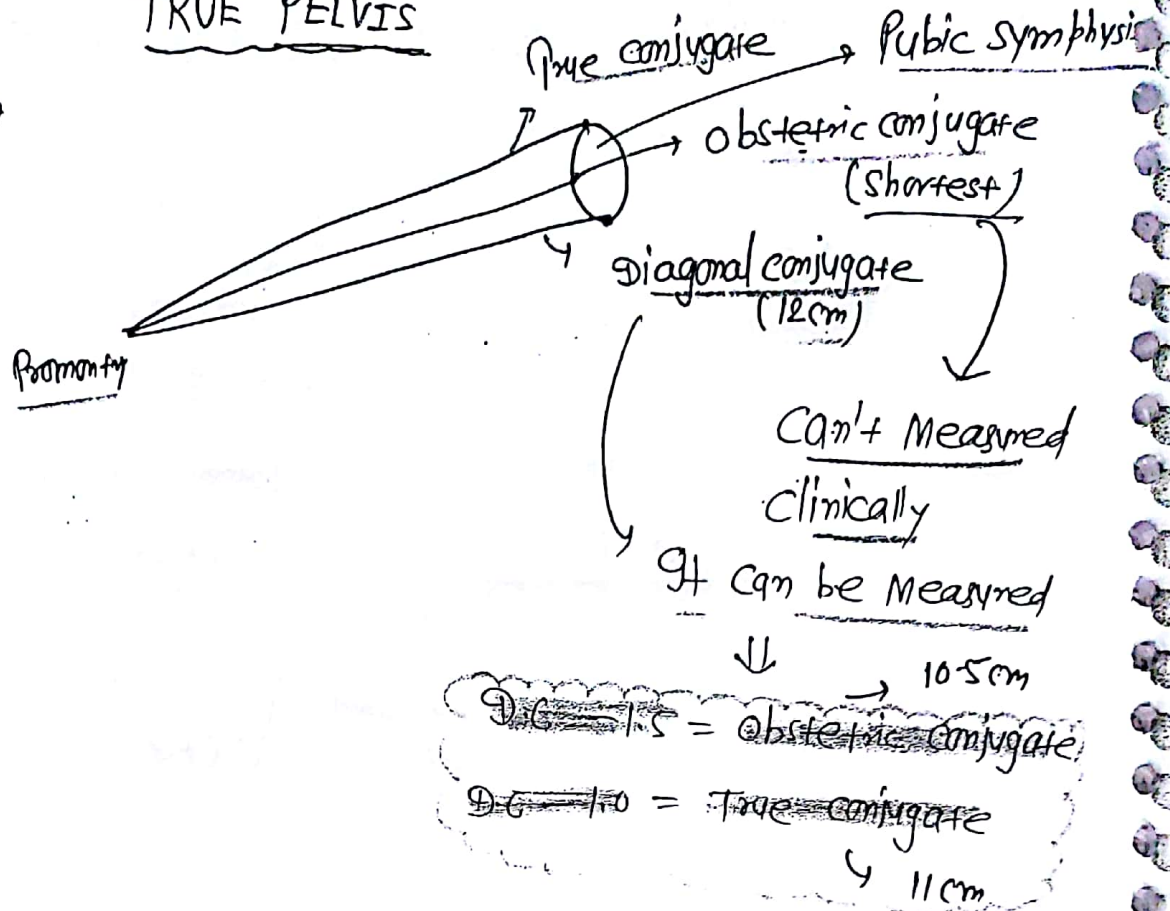
M/c of Occipito Posterior \Rightarrow Android Pelvis
(Posterior space is more than Anterior)SharpDeep transverse Arch (DTA)
L M/c in AndroidAcute

(85°)

Deep Pelvis(* Deepest Pelvis \Rightarrow Anthropoid)occipito posterior \Rightarrow AndroidPersistent occipito posterior \Rightarrow Anthropoid
Direct occipito posterior

TRUE PELVIS

INLET →



Cavity:

→ distance b/w ischial spine
Inter ischial spine diameter (IIS)

↳ 10.5cm

↳ we shouldn't able to touch the both ischial spine simultaneously Normally

Contracted cavity : $IIS < 8cm$

OUTLET →

Intertuberous diameter = 11cm
(ischial tuberosity)

↳ "Four knuckle test" is do to measure it.

Contracted Pelvis \Rightarrow Any 1 or More of the previously studied diameters are contracted (84)

\hookrightarrow Inlet $\Rightarrow < 10\text{cm}$ (for diagonal conjugate it is less than 11.5)
 Cavity $\Rightarrow < 8\text{cm}$
 Outlet $\Rightarrow < 8\text{cm}$
 \hookrightarrow do C.S.

to know about the contracted pelvis; we will do "Pelvic Assessment"

\downarrow
 In Primigravida; it is done @ 37 weeks
 In Multigravida; it is done when she goes in Labour

Q. G₂ P₁ L₁; Previous 1 C.S.; done for contracted Pelvis; comes @ 37 week ANC \rightarrow Recurrent Indication for C.S. (Never do VBAC)

* Cephalo Pelvic Disproportion (C.P.D.) \rightarrow

\hookrightarrow Best way to know \Rightarrow Trial of Labour

\downarrow
 MRI
 \downarrow
 CT Scan
 \downarrow
 X-Ray Pelvimetry
 \downarrow
 Pelvic Assessment

a G₂P₁L₁ ; Previous 1 CS done for C.P.D; she presents C
Labour & Pain 37 wk ; os 1 cm dilate ; 70% effaced vertex
at $\Rightarrow -3$; What is Mx?

do VBAC

↓

Trial for VBAC

CPD is Non-Recurrent Indication for CS

- * Plane of Least Pelvic dimensions = obstetric outlet
↑
Forms Roof of obs. outlet
= ischial spine
(Passes laterally to ischial spine)
- * Anatomic outlet = ischial tuberosities
(Passes laterally to ischial tuberosity)
- * Plane of Greatest Pelvic dimensions = Disc space b/w S₂-S₃

II. PUSH

Uterine contraction ; Pacemaker \Rightarrow Rt. cornu

Strongest \Rightarrow Fundus

Rate of speed \Rightarrow 2 cm/sec ; depolarise entire uterus in 15 sec

at what Intrauterine pressure
contraction became palpable

\Rightarrow 10 mm Hg

at what Gup contraction became
painful

\Rightarrow 15 mm Hg

\hookrightarrow Min Pressure Required to initiate
cx dilatation

* Fundus can't be intended \Rightarrow Moderate contraction

\Downarrow
40 mm Hg

(85)

* end of 1st stage \Rightarrow 50 mm Hg

2nd stage \Rightarrow 80 mm Hg

* Adequate uterine contraction \Rightarrow

i) 3 contraction in a span of 10 min & each is lasting for 45 sec;

ii) 16 contraction generates pressure of ~~220-250~~ Montevideo Unit.

Montevideo Units \Rightarrow No. of contraction in 10 min \times "P" generated

good or bad? \downarrow Bad
Tachysystole \Rightarrow > 5 contractions / 10 min
 \swarrow UPI \rightarrow Fetal distress

Tachysystole + Fetal distress

Hypersimulation

\rightarrow b/c Most of blood supply goes to baby in diastole

- Rx
- \rightarrow 1stly Stop the Infusion
 - \rightarrow Left Lateral Posture
 - \rightarrow O₂ by Mask
 - \rightarrow I/v fluids
 - \rightarrow Tocolysis (Stop the contraction)

* In Augmentation of Labour

Oxytocin given

Misoprostol acid

↳ May cause hyperstimulation
Rupture uterus

Management of

⇒ LABOUR

FALSE

No change in Cx dilatation

↓

Mx ⇒ Rest & Sedation

Amount of Cx dilatation ⇒

Latent Phase

0-5 cm

(0-3) WHO

↓

Rest & Sedation

↓

wait & watch

Inadequate Labour

↓

Mx ⇒ Augmentation

ARM (if low prostaglandin)
↓ after 30 min Oxytocin

TRUE

Progressive Cx dilatation

What stage

Active Phase

≥ 6 cm

↓

Is the Labour slow?

Yes (< 1 cm/hr)

No (1 cm/hr)

↓

Contractions are Adequate

or No?

No (Adequate Labour)

↓ CPD of Position

↓ do Release mechanism?

Moulding
caput (swelling of scalp)

↓ In slow labour
↳ make d
CPD

Q. Pt. comes @ 2 PM. 6 cm dilated Cx; 70% effaced;
Vertex @ -1; Membrane absent, Liquor clear, contracted.
220 mm (Montevideo Units) (Ruptured) (86)

↓
@ 6 PM; 7 cm 80% effaced; Vx @ -1; caput (+)
Moulding (+)

↓ do Trial of Labour

@ 10 PM; Finding absolutely same

↳ i.e Arrest of Labour

↳ do C.S.

OBSTRUCTED LABOUR

In General Physical Examination ⇒ Exhausted
dehydrated
Tachycardia
Tachypnea
Acidotic breath

On PLA examination ⇒ Upper segment ⇒ Tonically contracted

Lower uterine segment = stretched / thinned out

Blw U.S. & L.U.S. = depression (Ring)

Bandl's Ring / Pathological
Retraction Ri

Suprapubic bulge (Bladder)

FHR =

~~Severe distress~~

~~Absent~~

P/v examination =

~~Swollen Vagina~~

* If we put Foley's we can not be able to enter as it is

~~Edema / Moulding~~

Compressed by head we can Only able to put feeding tube
8 can see hematuria

~~Hematuria~~

Doesn't Pass Urine

is the earliest Marker of obstructed Labour

* obstruction is the absolute Indication for CS; even if the fetus is dead

* Destructive Procedures → Not in Modern obstetrics

↳ do craniotomy

Symphysiotomy

9/ Not done CS ⇒ Rupture Uterus

* if Patient present ~~Not passing urine~~ and told us about Bladder injury then we prove it as Not Bladder injury by

↳ VVF (vesicovaginal Fistula) in developing countries

↳ obstructed Labour

In developed countries

↳ Gynaecology (Malignancy)
surgery

III. Passenger

(71)

Fetus convert (N) into (ABN) Labour (87)

Vertex



(N)

CPD

Non vertex (ABN) Labour

Malpresentation



M/c ⇒ Breech

R/F ⇒ i) Multiparity;

ii) (ABN) Pelvis;

iii) Prematurity;

iv) Poly/oligohydramnios

v) GICA

vi) Placenta previa

* M/c Malposition

↳ OP (occipito posterior)

BREECH

M/c Cause ⇒ Prematurity

POG

Breech

Frank



25%

3/4



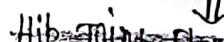
7-11%

Feet



3%

M/c type of breech ⇒ Frank breech (Extended breech)



Hip joint flexed, knee joint extended

Plv ⇒ we don't feel heel here

Complete breech (Flexed breech) ⇒ Least common type of breech.




Both knee & Hip are flexed; plv ⇒ -Gschial tuberosity; Anal opening; Heel Pad



* Primigravida \Rightarrow Frank breech

Multigravida \Rightarrow Complete breech

* Footling breech \Rightarrow Incomplete breech

Q  \hookrightarrow baby may have Cord prolapse
P/V \Rightarrow can see foot of baby

Cord prolapse \Rightarrow Exposed to temp.

\hookrightarrow Intense cord vasospasm
 \hookrightarrow death of baby.

Higher Risk in Footling breech

Less Risk in Frank breech

* Indication of C.S. in breech \Rightarrow

Absolute Indication \Rightarrow

① Footling breech;

② Staggers breech;

\hookrightarrow Hyperextended head

Relative Indication of C.S.

\hookrightarrow Vaginal is not C/I, but C.S. is preferred over vaginal delivery.

① Primigravida \bar{c} Breech;

aa ② Previous C.S. \bar{c} Breech;

③ Macrosomia \bar{c} Breech;

④ Hydrocephalus

\hookrightarrow do ventriculo-peritoneal shunt by Pediatric surgeon.

⑤ Prematurity

Q. Primigravida ; 37 week ; ANC checkup; o/E = Breech presentation; FHR (N); Liq (N); Placental fundal; Pelvis adequate; Mx = ? (88)

do ~~ECV (External Cephalic Version)~~
 can be done in Latent Phase of Labour
 ↳ for all ~~Non Cephalic presentation.~~
 if Requirements Met.

done for a) Single term ♀;

(b) ≥ 37 wk / 36 wk baby;

(c) Liquor Adequate;

(d) Membrane should be Intact;

(e) FHR (N)

(f) No C/I for vaginal delivery,

Relative c/I for ECV \Rightarrow Risk More than Benefit

↳ avoid ECV \Rightarrow (a) Previous CS

(b) GUR

(c) Pre-eclampsia

* ECV is always done under continuous fetal Monitoring;

* It is done under tocolysis (terbutaline if m);

* during ECV \rightarrow if fetal distress \oplus

↓
 Return baby to original position
 ↳ do CS.

Q. G₂P₁, L₁ 39 weeks Breech presentation



first try ECV



but if any C/I of ECV ⊕

→ Primigravida ⇒ C.S.

→ Multigravida ⇒ vaginal delivery
(Assisted) ~~Breech~~
vaginal delivery

if fetus has extended Arms ⇒ Lovset Manoeuvres

extended Legs ⇒ Pinard's Manoeuvres

After coming head ⇒ Burn-Marrall
Manoeuvres



Grab foot &
goes 180° towards
Pelvis of Mom;
after head comes out
by

spine of fetus is
& towards
Anterior the
obstetricians

↓
In this delivery of head by
"Flexion"; Not by extension.

After coming head ⇒ Mauriceau Smellie
vict (MSV)

* Burn Marshall & MSV

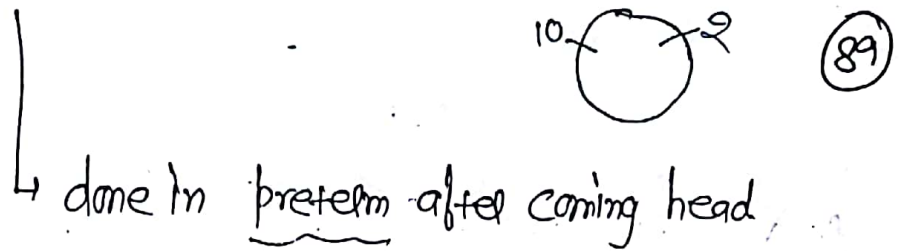


only when baby is DorsoAnterior
DorsoAnterior

↓
do Mallet bone flexion
& Shoulder traction &
delivery of head by "Flexion"

In Dorso-posterior breech; after coming head ⇒ Prague's Manoeuvres

* Dührssen's Incision \Rightarrow 2 Small Incision on Cx



\Rightarrow if most of the part of baby is delivered & only head is inside

* Forceps delivery \Rightarrow Piper's Maneuver

* Last Resort \Rightarrow Zavanelli Maneuver

* M/c cause of Fetal death \Rightarrow Cerebral Hemorrhage
in breech (ICH)

* O.P. (Occipito Posterior) \Rightarrow Not Malpresentation; Malposition

\Rightarrow M/c cause \Rightarrow Android Pelvis

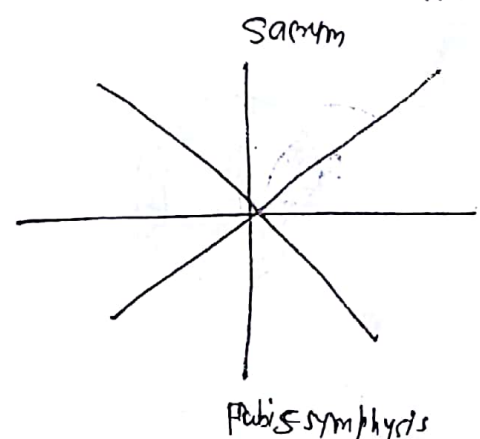
2nd M/c cause \Rightarrow Deflexed head



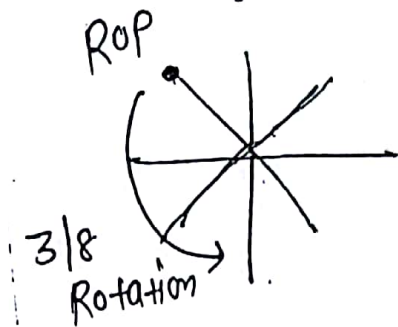
Engaged diameter \Rightarrow occipito/mental
41.5

M/c in Primigravida

M/c Position - R.O.P.



1st outcome \Rightarrow all favourable — Push
 — Passengers
 — Passage



\hookrightarrow so take much time; Slow progress of labour.



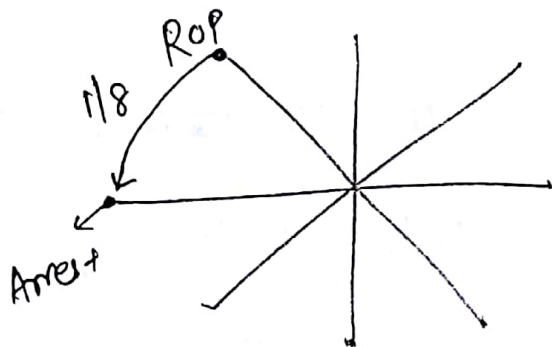
Rx \hookrightarrow wait & watch ; when baby is Anterior then delivers

2nd outcome \Rightarrow Deep transverse arrest

\hookrightarrow d/t android pelvis



do the C.S.



but if Pelvis is (N); then

i) if Inadequate contraction seen

\hookrightarrow give oxytocin

ii) if Contraction is Adequate

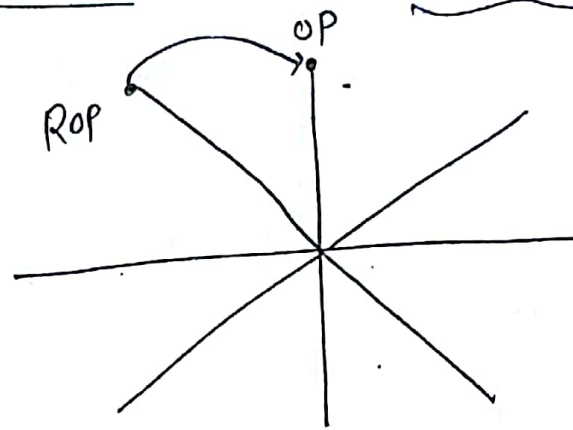
\hookrightarrow Manual Rotation

Forceps Rotation

Vacuum device extraction

3rd outcome $\frac{1}{2}$

Persistent OP / Direct OP

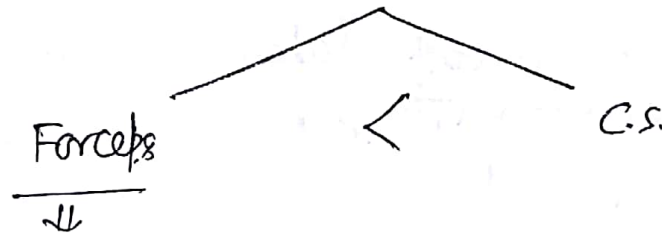


↳ seen in Anthropoid Pelvis
(90°)

⇓
Mechⁿ of Labour

⇓
"Face to Pubes" delivery

* In case spontaneous delivery (Not happening)



In Posterior Position \Rightarrow Incidence of Maternal Injuries & severity ↑

* f. of babies are in O.P. at onset of Labour = 20%

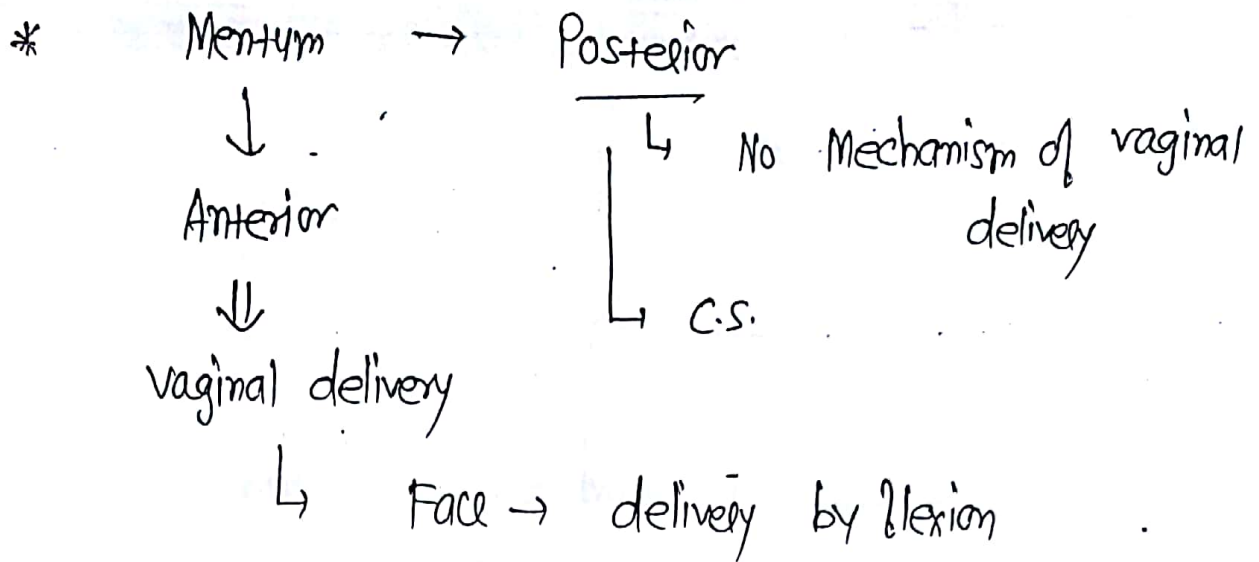
at the end of Labour = 5%

* FACE / BROW PRESENTATION

M/c cause of Face presentation \Rightarrow Anencephaly;

Pelvis type; which favour it \Rightarrow Platypelloid

Anything that prevents flexion of head \Rightarrow Risk factors (> 1 Loop of cord around Neck; tight loops /



- * Mentoposterior in Labour ⇒ C.S.
- Mentoposterior in early Labour ⇒ wait & watch
 ↳ to Rotate gives time

- * Brow presentation
- ```

graph TD
 A[Brow presentation] --> B[engaging Diameter ⇒ Mentoverical = 14cm]
 A --> C[No Mechanism of Labour]
 A --> D[Brow in Labour → C.S.]
 D --> E[Brow in early Labour → wait & watch]

```

### TRANSVERSE LIE

M/C cause ⇒ Prematurity

M/C cause at term ⇒ Placenta previa

- Most Commonly Seen in "Platyphelloid" pelvis.
- Highest Risk of cord prolapse

\* Transverse Lie — In Labour — do C.S.

↳ Tries ECV  
if Requirement Met. (91)

\* Neglected Shoulder

↳ Upper segment  $\Rightarrow$  Tonically contracted

Lower segment  $\Rightarrow$  Stretch

Bend's Ring  $\oplus$  in b/w us & LUS



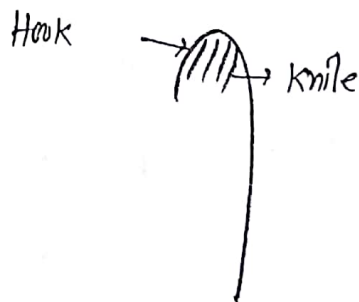
Case of obstructed Labour

↳ FHR Absent

↳ do C.S.

Destructive procedure  $\Rightarrow$  i) Evisceration;

ii) Decapitation;



Q. Lady pr.  $\bar{c}$  Neglected shoulder presentation  $\bar{c}$  dead baby.  
all the following can be done except  $\Rightarrow$

a) C.S.

b) Evisceration

c) ~~craniotomy~~ (b/c for it vertex presentation Required)

d) Decapitation here transverse lie pr.



Q:

Confusion of Breech presentation on p/v examination  $\Rightarrow$

### Face presentation

- $\hookrightarrow$  Frank breech Most commonly confuse.
- $\hookrightarrow$  In it ~~three~~<sup>two</sup> bony prominence & one opening (Mouth) Makes a triangle; while in breech @ it forms straight line

### INSTRUMENTAL DELIVERY

- do when we cut short 2nd stage of Labour
- Pre-Requisites  $\Rightarrow$

- F  $\rightarrow$  Fully dilated Cx
- O  $\rightarrow$  No Obstruction in the path
- R  $\rightarrow$  Ruptured Membrane
- C  $\rightarrow$  Good uterine contraction
- E  $\rightarrow$  Engaged head / Empty bladder / episiotomy
- P  $\rightarrow$  Favourable presentation

## Forceps delivery

- difficult
- Maternal Exhaustion
- we're heart disease
- In Fetal distress (Forceps > Vacuum delivery)
- \* In general Forceps prefer over Vacuum
- In pre-term
- Face presentation (MentoAnterior Position)
- After coming head of breech (Piper's Forceps)
- More Maternal Injuries

## Vacuum delivery

- easier Application (92)
- Needs Maternal effort
- also we're heart disease; Not C/I
- if POG < 34 weeks, Vacuum is absolutely C/I
- In Face presentation C/I.
- More fetal Injuries

Q Which fetal Injuries are M/C in Forceps delivery??

- IVH
- Facial Nerve Injuries
- Brachial Plexus Injuries
- Cornea of the eye

Q Injuries which see in Vacuum Not in Forceps delivery??

- 6th Nerve Injuries
- Cephalhematoma
- Retina Injuries

# \* Classification of Forceps delivery $\Rightarrow$

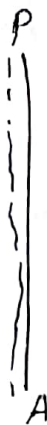
Outlet  $\geq +3$

Low  $+2$

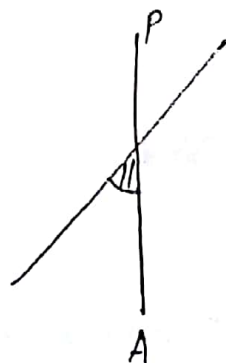
Mid cavity - b/w 0. & +2 } Not in Modern days  
High - above 0

## \* criteria for application of outlet forceps $\Rightarrow$

- ① Scalp visible at Introitus
  - ② head on Pelvic floor
  - ③ Skull on Perineum
- $\geq +3$
- ④ Sagittal Suture should be preferably in A-P Position



⑤



Rotational defect  $< 45^\circ$

$\hookrightarrow$  apply outlet forceps

How to know that Forceps is correctly applied <sup>183</sup> ⇒

⇓

(93)

Blades have to be Equidistant from sagittal Suture

Along which fetal diameter we apply Forceps blade

↳ Occipito Mental (13.5 cm)

\* Left blade of Forceps ⇒ Introduced first

↳ M/c Position = Left,

\* M/c outlet forceps used ⇒ Wrigley's outlet forceps

↳ Not used in After coming head; b/c of short nature

⇓

for both outlet & Low position,  
short forceps & have English Lock

\* Kelland's forceps ⇒ for Rotational defect

↳ Long forceps

\* Piper's forceps ⇒ for after coming head

↳ Pelvical curve ⊕ (Baby body Rest here)  
very long forceps



## VACUUM DEVICES

- Use only plastic cup

↳ Bell shaped

diameter = 5-7 cm

Silastic

Pressure =  $0.8 \text{ kg/cm}^2$   
Generated

centre of the cup @ Flexion Point

↓

3 cm Anterior to Posterior fontanel  
OR

6 cm Posterior to Anterior fontanel

@ sagittal suture

- The Margin of cup touches the posterior fontanelle

Failed Vacuum  $\Rightarrow$  3 Pulls - No descent of head  
3 Pop offs

\* if one device fails we go for C.S.

CI of Instrumental delivery  $\Rightarrow$

- a) Contracted Pelvis
- b) CPD.
- c) HIV+ve Patient
- d) K/clo coagulation defect
- e) osteogenesis imperfecta

} in babies

# ANTEPARTUM HEMORRHAGE (APH) (94)

\* Bleeding from or into the genital tract beyond the period of viability

↳ In India  $\approx$  28 weeks

\* Causes of APH  $\Rightarrow$  Abruption

Placenta Previa

↳ Placenta Lying in the LUS

↳ Premature separation of a Normally Located Placenta (from Underlying decidua)

## ABRUPTION

Advanced Maternal Age

Smoking

Multiparity

Highest Risk

Previous history upto 1st.  
(Relative Risk)

Pre-eclampsia ; Thrombophilia      High Risk  
Trauma      Folic acid deficiency  
Polyhydramnios      Fibroid  
PROM  
Long standing oligohydramnios

Early  $\oplus$  USG

↳ In subsequent scan  $\Rightarrow$  Placenta  $\Rightarrow$  Upper segment

In previous c.s.

Migration abt

↳ So: RIF for Placenta previa

## PLACENTA PREVIA

Previous history  
(5+)

Previous L-S.C.s  
(More the Number higher Risk)

Placenta May be Low Lying

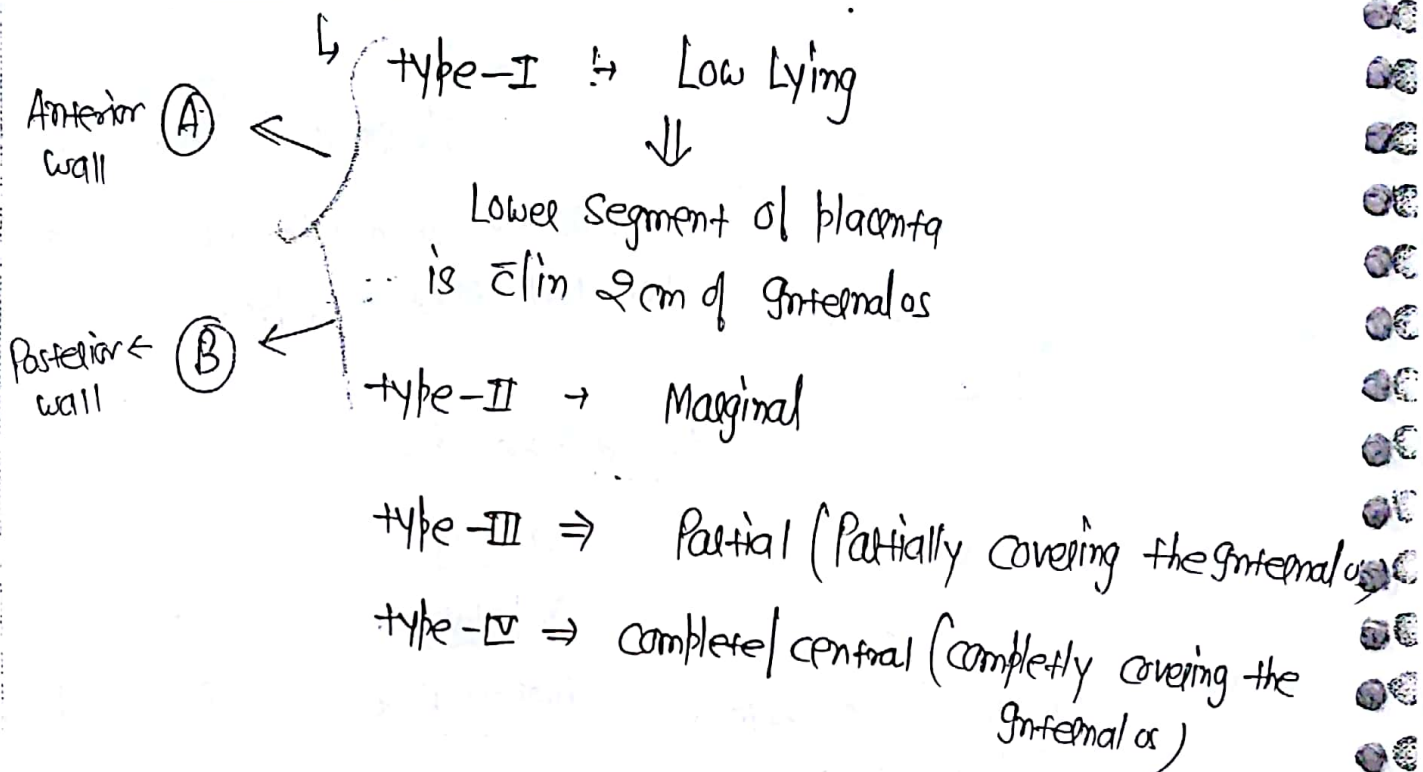
d/t differential growth of uterus  
Placenta migrates

Endometritis

Mullerian Anomalies

## CLASSIFICATION

### Placenta PREVIA



Other classification ⇒ Mode of delivery ⇒ Vagina /

Minor Placenta previa ⇒ Type 1A; 1B; 2A

Major " " ⇒ Type 2B; 3; 4

↳ Mode of delivery ⇒ C-S

\* Dangerous Placenta previa ⇒ 2B ⇒ Fetal distress

↳ On type 2B ← Stallworthy sign ⇒ Pushing down of head produces a dip in FHR.



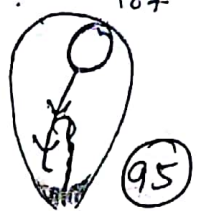
Type I



Type II



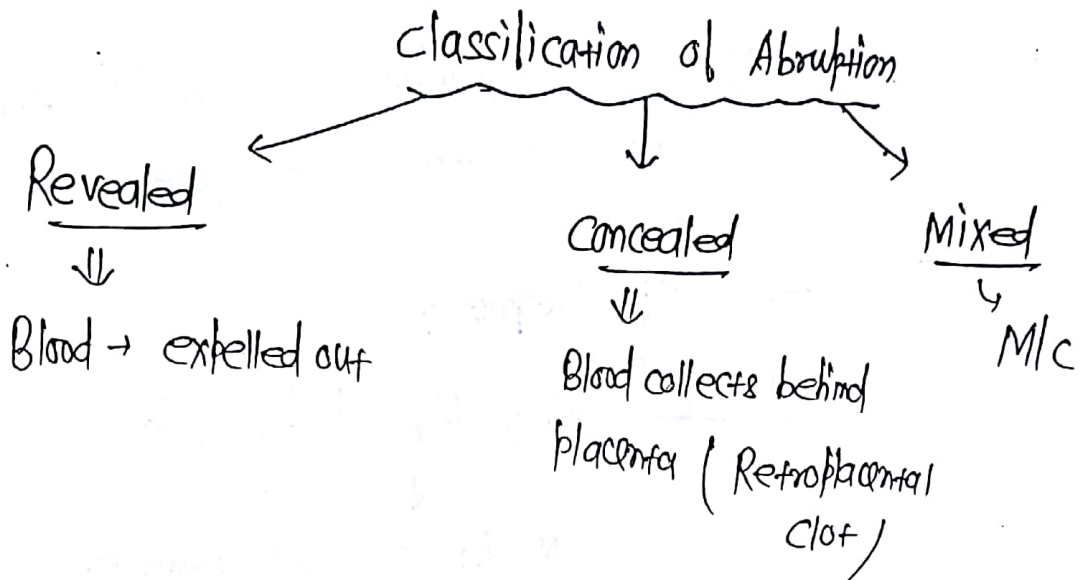
Type III



Type IV

187

95



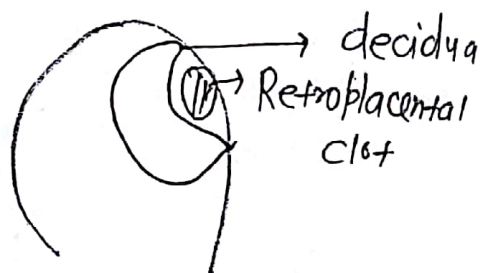
\* PAGE'S classification of Abruption ⇒

Type 0 ⇒ Retrospective diagnosis,

Type 1 ⇒ bleeding & Pain (FHR ⇒ (N))

Type 2 ⇒ Bleeding + Pain + Fetal distress

Type 3 ⇒ Bleeding + Pain + GUD + Shock  
± DIC  
Maternal

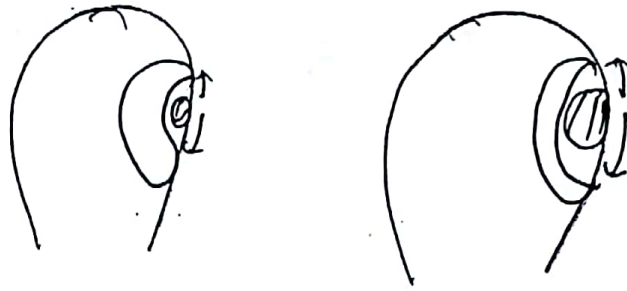


⇒ tissue thromboplastin

↓  
Potent uterotonic  
(Pain)



\* Abruptio is self propagating



Risk of DIC is dependent on how long Abruptio to delivery times taken

\* Termination of Pregnancy

↳ only Mx of Abruptio



No Role of Conservative Mx.

\* Pt. Comes w/ APH; how to tell about P.P. & Abruptio?

P.P.

Bleeding

↳ Painless

Bright Red (Fresh bleeding)

causeless

M/c Presentation

Hypotension M/c

⇐ General Physical examination

Abruptio

Bleeding

↳ Painful

→ Altered (dark coloured)

→ Preceding event

↳ Pre-eclampsia

Trauma

⇒ Hypotension is Not common (b/c base BP is high)

P.P.  
 Uterus Relaxed  
 Non-tense  
 Non-tender

Fundal height = POG  
 < POG (Some-times)

P/A  
examination

Abruption  
 Uterus tense  
 tender (96)

Fundal height > P.O.G.

Less common

Fetal  
Distress

More Common

Most Common  
 (Fundal height < POG)  
 Transverse Lie

Malpresentation

Less common

H/o warning hemorrhages

No warning hemorrhage  
 (Acute event)

Should not be done in APH  
 Until you Rule out placenta previa.

P/s And  
 P/v examn

Transabdominal USG

IOC

\* TVS is Not C/I; but it  
 is More Sensitive for Posteriorly  
 Located Placenta.

Qe

Pt. Comes to APH

↳ Pt. Not Low Lying (Fundal)

No RPC

ans:

Still a case of Abruptio

↳ Plv examination

↳ ARM

Confirm

↓

Blood stained  
Liquor

Induce Labour

\*

Placenta praevia

↳ Mx ⇒ Are there any indications for TOP

↓

Yes (It is in order to do TOP)

↓

i) P.P. + Unstable vitals; (M/c Indication for TOP)

ii) P.P. + Fetal distress;

iii) P.P. + > 37 weeks.

iv) P.P. + Continuous Bleeding Per vagina

v) P.P. + Woman goes into Active Labour

vi) P.P. + GUD

vii) P.P. + GEA (Incompatible for Life)

do it Right Now

Q.  $G_{13}P_2L_2$ ; 34 weeks APH (Bleeding p/v; Painless); on examination Fundal height = 34 weeks; Relaxed Non-tender Non-tense; Relaxed uterus; RR = 100/m; BP = 114/76. (97)

USG = type 4 PP.

$M_x \Rightarrow$  Conservative  $M_x$ .

- \* Indication of CS in APH  $\Rightarrow$
- i) Unstable vitals;
  - ii) Fetal distress;
  - iii) Major degree of Placenta previa;
  - iv) Full-term ( $< 32$  wk)

\* Conservative  $M_x$  in Placenta previa

$\hookrightarrow$  Macaffee Regimen

i) Bed Rest

ii) Maternal Monitoring : vitals;

only bleeding is not  
indication of blood transfusion;  
Anemia also a indication

FH (Fundal height)

AC (Abdominal circumference)

BPV (Bleeding Per vagina)

Baseline Ix

uterine contraction

iii) Fetal Monitoring : FHR

iv) if patient  $< 34$  weeks  $\Rightarrow$  Steroids for Fetal Lung Maturity

Dexamethasone (National guideline)  $\Rightarrow$  Doc

$\Downarrow$   
6 mg  $\cdot$   $\frac{1}{12}$  hr  $\times$  4 doses  $\times$  12 hr apart



Beta Methasone - 12 mg x 1/m x 2 doses 24 hrs apart.

v) < 34 wk + Uterine Irritable (Mild contraction)

↳ give Tocolytics (to buy time for steroids cover)

↳ also for Ecr

Hyperstimulation

• Doc (Tocolytics) ⇒ Nifedipine

• Safest Tocolytic ⇒ "

• Doc of Tocolytics in heart d. ⇒ Atosiban (Oxytocin Receptor Antagonist)

• Initially β-Agonists used as tocolytics

↳ cause ⇒ Hyperglycemia AIIMS May 18

Hypokalemia

Tremors

Tachycardia

Amythmia

Cardiac arrest

Pulmonary edema

only β-Agonist used as Tocolytic

Now a days

↓

Terbutaline

Indomethacin

↳ Not given > 32 week

↳ Premature closure of ductus arteriosus

Halothane

Alcohol

Diazoxide

MgSO<sub>4</sub>

(acts as tocolytic @ 9-10 meq.)

↳ given 4-7 meq

It has Neuroprotective action (prevent cerebral palsy) for preterm babies

↳ toxic dose for Mother

so Not given as a tocolytic agent.

## End point of conservative Mx

(98)

↳ 37 wks @ or any other indication of TOP.

ABRUPTION ⇒ No conservative Mx

↳ Indication for TOP  
 ↳ No Role of Tocolysis  
 ↳ Vaginal delivery is preferred if  
 ↓  
 ble she has high Risked DIC  
 &  
 Progress very quickly in Labour  
 (tissue thromboplastin)  
 Precipitate Labour (entire process of Labour in  
 3 hr)  
 ↳ do cs; when ⇒ Unstable vitals  
 Fetal distress  
 Far from term

ii Intraoperatively we see Red Uterus

↓  
 COUVELAIRE UTERUS  
 (Bleeding in Myometrium)  
 Seen in Uteroplacental Apoplexy / Abruptio.

- ↓
- It is Not an Indication of Hysterectomy
  - It is R/F for PPH.

Q. 34 week G; Bleeding Plv & Pain; O/E uterus 36 wk; tense, tender; FHR = 100 bpm; on Evaluation BT/CR/INR = deranged  
Mx  $\Rightarrow$  It is the case DIC

Transfuse FFP and try for vaginal delivery.  
In Cesarean section; Pt. Bleed profusely from all the sites & dies on table.

Most cause of DIC in obstetrical  $\Rightarrow$  Abruptio.

\*

Placenta previa  $\rightarrow$  term



Mx  $\Rightarrow$  USG

OR

do "Double set up examination"

$\swarrow$  Plv in O.T  $\searrow$

1 table



Ready for emergency C.S.

2nd table



On Plv  $\Rightarrow$  Fornices bogging  
( $\geq 2$  fornices)



We are done for Major placenta previa

$\swarrow$   
if we don't get any fornices bogging



Goes up to the level of Internal os



if bleeding (+)

or Placental tissue (+)  
Near Internal os.



do emergency C.S.

if No placental tissue

Near the Internal os  
(Minor)



ARM  $\rightarrow$  Labour Room

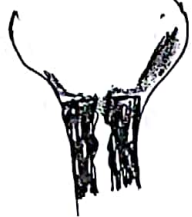


# ADHERENT PLACENTA

(99)

Placenta is attached to Decidua; but here Placenta is attached to Myometrium & Intervening decidua is ~~absent~~

- Types  $\Rightarrow$  ① Accreta  $\Rightarrow$  Attached to Myometrium  
 $\hookrightarrow$  Most common  
 $\hookrightarrow$  "Large Intraplacental Lacunae"  $\Rightarrow$  Sonological finding



② Increta  $\Rightarrow$  Invades into the Myometrium



③ Percreta  $\Rightarrow$  Penetrates through & through the Myometrium. It comes out in the serosal surface.  
 $\hookrightarrow$  Least common



- Highest R/F  $\Rightarrow$  Previous L.S.G.s. + Placenta previa (current)

QA Highest R/F for Adherent Placenta  $\Rightarrow$

④ Previous L.S.G.s.

⑤ Previous P.P.

Other R/F  $\Rightarrow$  Curettage  
 Scar  
 Multifarity  
 Increasing Age

IOC  $\Rightarrow$  USG + Doppler

Heterogenous Placenta  
 $\hookrightarrow$  Intraplacental Lakes  
 (Blood tissue in placental tissue)



if we have any doubt / OR we <sup>want to</sup> know about depth

↓  
do MPR

(N) prt. b/w placenta & decidua

on HRE ⇒ Absent / Incompletely developed Mitabuch's Membrane (Layer)

↓  
It is fibrinoid degeneration  
b/w trophoblast & decidua

Mx ⇒ Kleio Adherent Placenta

↳ Elective CS (classical) \*\*

+  
Hysterectomy

↳ if we incise on LUS;  
Myometrium cuts & we  
damage placental tissue

• presentation in Undiagnosed case ⇒  
deliver

↳ No Sign of Placental separation

↓  
if we try to Manual Removal

↓  
Pt. bleeds

↳ Refractory PPH (Pt. pr. with Refractory PPH)

↓  
do hysterectomy

\* if Patient denies hysterectomy

It will go autolytic digestion

c/m 6 months

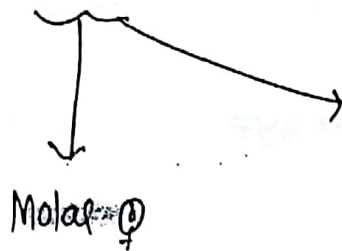
↳ deliver vaginally

↳ cut the cord short as possible as close  
to placental end

leave the placenta  
in situ

# MOLAR PREGNANCY

GTD  $\Rightarrow$  Gestational Trophoblastic disease



- Molar  $\Rightarrow$
- Partial
  - Complete

G.T. Neoplasia

- Invasive Mole
- Choriocarcinoma
- PSTT (Placental site trophoblastic tumor)
- ETT (Epitheloid trophoblastic tumor)

## PARTIAL MOLE

Chromosomal Make up  
of Molar  $\Rightarrow$

$\hookrightarrow$  Triploid (9at)  
 $\hookrightarrow$  69 XXY

10+ Tetraploid  
Extra set - Paternal  
Dispermic

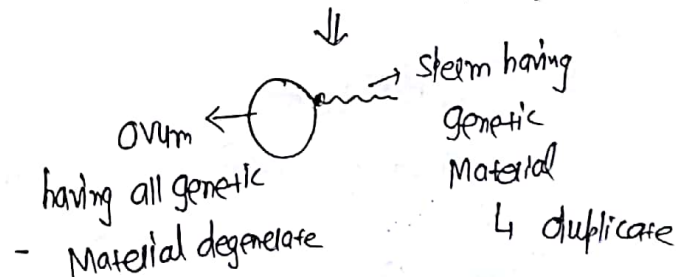


## COMPLETE MOLE

Diploid (46XX)  $\xrightarrow{9at}$

Monospermic (10+ 46XY)  $\xrightarrow{10+ 46XY}$   
(Dispermic)

All genetic material is  
Paternal



Bleeding  
(2nd Trimester  $\Rightarrow$ )

O/E  $\Rightarrow$  Fundal height  $\leq$  POG

Miscarriage USG

$\hookrightarrow$  Missed Abortion

M/c presentation

Bleeding (2nd Trimester  $\Rightarrow$ )  
Like vesicle

O/E  $\Rightarrow$  Fundal height  $>$  POG

USG

$\hookrightarrow$  Snow storm appearance

## PARTIAL MOLE

- Fetus Seen
  - ↳ dies d/t Multiple Anomalies

\*If we can see local hydropic in Placenta

Gest sac  $\Rightarrow$  Transverse diameter  $>$  AP diameter

Molar  $\oplus$  Most commonly diagnose in 1st Trimester (also in 1st trimester)

- HCG  $>$  than expected (but Not Markedly high)

HCG

- Markedly Raised ( $>105$ )

HPE

— Gold Standard —

- Less Marked trophoblastic Proliferation (Confirmation of)
- Focal hydropic degeneration of villi
- Fetus  $\oplus$
- Vascular

HPE

- extensive trophoblastic Proliferation;
- complete hydropic degeneration of villi
- No Fetus
- Avascular

- Trophoblastic

Scalloping  $\oplus$

$\downarrow$

Stromal Inclusion

X

X

X

X

Medical complication

$\rightarrow$  d/t high hcg  
i) Thyroid Storm

ii) Pulmonary embolism

iii) Early onset pre-eclampsia  
↳ d/t paternally exposed Ag.

iv) Hyperemesis gravidarum  
↳ d/t hcg

## COMPLETE MOLE

• No Fetus on USG Seen

• ovaries  $\rightarrow$  Theca-lutein cyst in it.

↳ 97-98-99 cases  $\approx$



Partial  
Partial mole  $\rightarrow$  GTN (3-5+)

Complete <sup>199</sup>  
Complete mole  $\rightarrow$  GTN (15-20+)

Partial mole  $\rightarrow$  chorio. ( $< 1+$ )

Complete mole  $\rightarrow$  chorio. (4+)

$M_x \Rightarrow$  ~~Suction & evacuation (do till Fetal height  $\leq 12$  wks)~~

$\Downarrow$

if pt. is  $> 40$  yr + Complete Mole  $\oplus$

$\hookrightarrow$  do Hysterectomy ( $\downarrow$  Risk of chorio-ca)

$\rightarrow$  S/E  $\Rightarrow$  Pulmonary Embolism

Thyroid storm

to prevent Embolism  $\Rightarrow$  Start suctioning first then followed by oxytocin.

Check curettage  $\Rightarrow$  Sharp

\* Send sample for HPE

$\downarrow$

then follow up  $\bar{c}$  HCG

Weekly s. HCG  $\Rightarrow$  until 3  $\textcircled{N}$  values

$\Downarrow$

Monthly HCG

Total  $\Rightarrow$  up to 6 Months  
Surveillance

\* Avg. time to  $\beta$ -hcg to become  $\textcircled{N}$   $\Rightarrow$  9 weeks

Avg. time to  $\beta$ -hcg to become  $\textcircled{N}$  in Partial mole  $\Rightarrow$  7 weeks

\* Say patient Not to conceive

gud Avoid  
bleed bleeding

$\hookrightarrow$  Contraceptive of choice  $\rightarrow$  OCP  
Barrier can be used



\* CRITERIA FOR Diagnosis of GTN (Any 1) :-

- ① 4 consecutive HCG values shall show of Plateau  
(Less than  $\pm 10\%$  values)  
 $D_1 - 7 - 14 - 21 (\pm 10\%)$
- ② 3 consecutive HCG value that shows a Rise  
 $D_1 - 7 - 14 \text{ rise } (>10\%)$
- ③ HCG Remains elevated even after 6 months of Suction & evacuation
- ④ HRE  $\rightarrow$  GTN

\* Clinical presentation of GTN :-

- i> Bleeding Per vagina — Persistent
- ii> Shock
- iii> Persistent Theca Lutein cyst

$\downarrow$

④ Theca Lutein cyst shows Spontaneous Resolution  
in 2-4 months of Suction & evacuation.

iv Uterine Subinvolution

$\hookrightarrow$  uterus doesn't go to ④ Non-pregnant state after Suction & evacuation

v Metastasis

\* High Risk for conversion of GTN  $\Rightarrow$

- i) ~~≥ 40 yr~~ (Maternal Age)
- ii) Fundal height  $> 90$ cm
- iii) HCG  $> 105$
- iv) Blc Large ( $> 6$ cm) theca Lentin cyst

Should Receive  
Prophylactically  
Chemotherapy  
 $\downarrow$   
Actinomycin D  
OR  
Methotrexate

\* M/c GTN  $\Rightarrow$  Invasive Mole

Lc GTN  $\Rightarrow$  ETT

M/c GTN to develop after Full term @  $\Rightarrow$  Choriocarcinoma

\* Choriocarcinoma M/c develop after  $\Rightarrow$  Complete Mole  
which type of @

Malignant



Metastasis seen (Common ball Metastasis on CXR)

L M/c site  $\Rightarrow$  Lungs  $>$  vagina  $>$  Liver  $>$  Brain

2nd M/c finding on Choriocarcinoma  $\Rightarrow$  "Snow Storm Appearance" on CXR Lung

$\downarrow$   
also seen in USG of complete mole

\* In vagina

L Bluish Suburethral Nodules (+)

L don't take biopsy b/c very vascular

\* How to differentiate Invasive Mole from Choriocarcinoma

(HPE)  $\Rightarrow$  Presence of villi  $\Rightarrow$  Invasive Mole  
 Absent villi  $\Rightarrow$  Choriocarcinoma

Choriocarcinoma

- M/c
- M/c after complete Mole

Tumor Marker  $\Rightarrow$  HCG

Malignant

~~Anaplastic~~ cells

(Cyto syncytial trophoblast)

• Hemorrhage / Necrosis +

• Chemo sensitive

• TOC  $\Rightarrow$  Chemo-therapy

PSTT

Rare

M/c after ETP

Full term  $\phi$

HPE  $\Rightarrow$  PLAP

Human Placental Lactogen

Not Benign

Placental alkaline Phosphate

Mononuclear Monomorphic cells  
 (Not syncytial ones)



Chemo Resistant

Hysterectomy

\* STAGING OF GTN  $\Rightarrow$

Stage I  $\Rightarrow$  Tumor is confined to uterus

Stage II  $\Rightarrow$  - Outside uterus but in pelvis

Stage III  $\Rightarrow$  Metastasis to Lungs (Good Prognosis)

Metastasis to lungs

Stage IV  $\Rightarrow$  Metastasis elsewhere except + vagina

PHET

Modified WHO Scoring  $\Rightarrow$  Low Score  
 (Good prognosis)

High Score  
 (Bad prognosis)

i) Age  $\leq 39$  yr

$\geq 40$  yr

ii) HCG ( $\leq 40,000$ )  $\leq 10^3$

$\geq 10^3$



|                                                 |                               |                               |
|-------------------------------------------------|-------------------------------|-------------------------------|
| iii) Type of Antecedent                         | Molar                         | FITP (Full term Pregnancy)    |
| iv) <del>Duration of Antecedent pregnancy</del> | <del>Less than 4 months</del> | <del>More than 4 months</del> |
| v) Site of Metastasis                           | Lungs                         | Liver, Brain                  |
| vi) No. of Metastasis                           | < 4                           | > 8                           |
| vii) Tumor size                                 | < 3 cm                        | > 5 cm                        |
| viii) H/o Previous Chemotherapy                 | Single Agent                  | Multi Agent                   |

if Total WHO Score  $\leq 6$

↳ Low Risk GTN



→ Single agent chemotherapy



Methotrexate

↓ if Pt. Resistant

Actinomycin D

also in stage I

$\geq 7$

High Risk GTN



Multi Agent chemotherapy

E → Etoposide — D<sub>1</sub>

M → Methotrexate — D<sub>2</sub>

A → Actinomycin D — D<sub>3</sub>

C → Cyclophosphamide — D<sub>4</sub>

O → Oncovin (vinorelbine) — D<sub>5</sub>

↓ if Resistant

E → D<sub>1</sub>

M → D<sub>2</sub>

A → D<sub>3</sub>

E → Etoposide

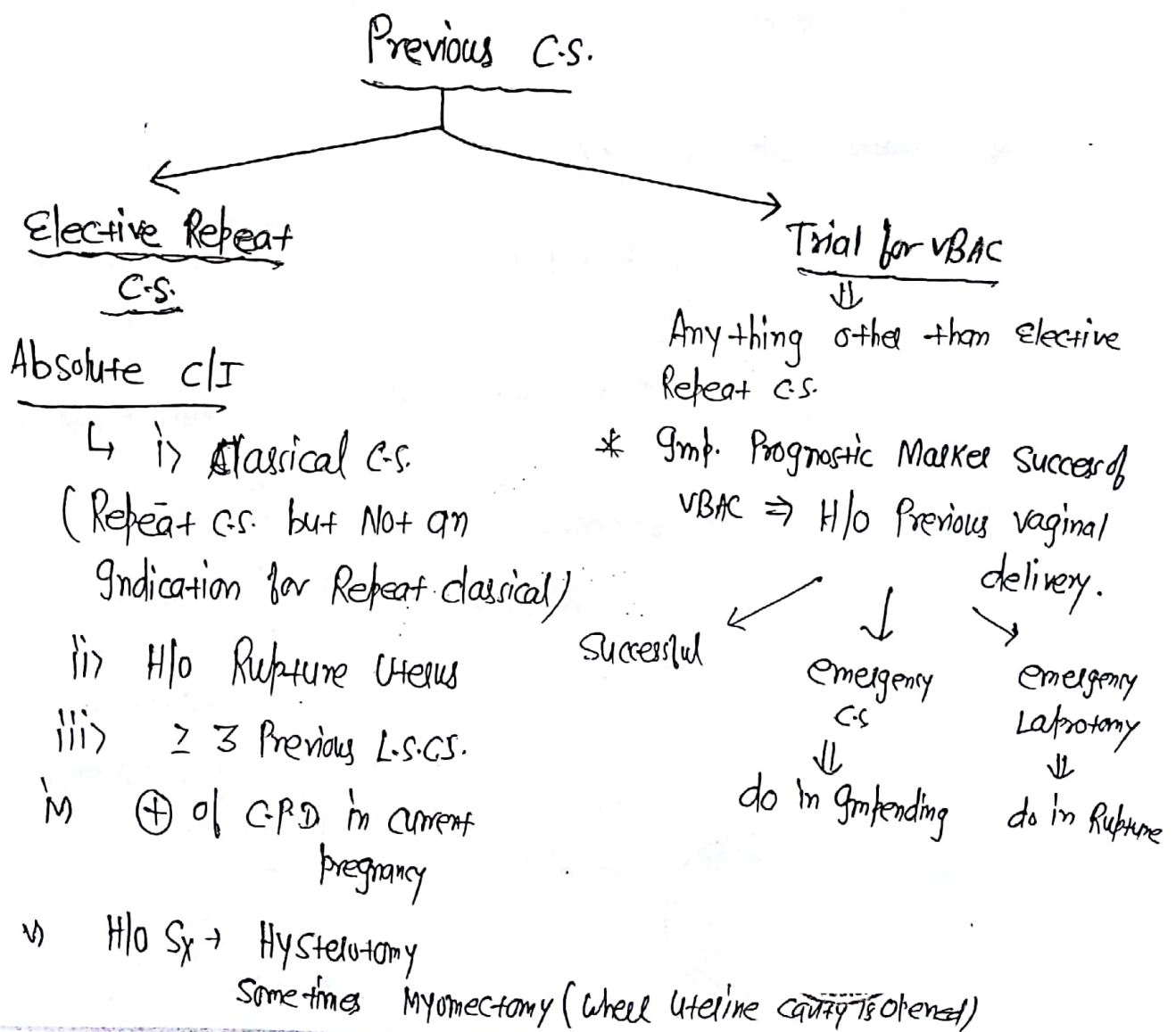
P → cisplatin



Follow up  $\Rightarrow$  Weekly hcg - till 3<sup>rd</sup> (N) value  
 End point of chemo.  $\Rightarrow$  1st (N) value - 3 More cycles of chemotherapy  
 After 3<sup>rd</sup> (N) value  $\Rightarrow$  Monthly follow up

Period of Surveillance for Low Risk GTM - 12 Month  
 " for high " "  $\rightarrow$  24 Month

Advice & Contraception of choice is same as Molar  $\phi$   
 $\Downarrow$  Combined ocp's  
 Not to conceive



Q.  $G_{12} P_{1L}$  = previous L.S.Gs.; done for C.P.D. 40 wk; Head is breech bloating Cx os closed <sup>unellaced</sup> My

(104)



C.S. (b/c C.P.D. ⊕ in current ⊕)

vi) where vaginal delivery is CI (Contracted Pelvis  
Major degree of Placenta previa)

\* Relative Indication for Repeat C.S. (C.S. > V.D.) :-

i) Previous L.S.Gs. — ⊕ Breech in current ⊕

ii) " — ⊕ Macrosomia

iii) " — ⊕ Post-term

\* Sign & Symptom of Impending Rupture :-

i) Non Reassuring FHR

↳ 1st FHR changes ⇒ Tachycardia

M/c FHR changes ⇒ Bradycardia

ii) evidence of Maternal Tachycardia;

iii) evidence of Scal tenderness (Pain);

become only significant if Non-Reassuring FHR ⊕.



if Intraoperatively; at the Scal site — all Layer of Myometrium have given away but overlying Serosa intact



Very Imp. for Next ⊕

← "Scal Dehiscence"

## \* Sign & Symptoms of Rupture of Uterus →

- i) Maternal Tachycardia;
- ii)  $\pm$  Hypotension;
- iii) Severe fetal distress / FHR absent;
- iv) Fetal parts are superficially felt; (as the fetus is expelled into Abdominal cavity)
- v) Uterine contour is lost.
- vi) Sudden stoppage of uterine contraction;
- vii) Fresh bleeding per vagina;
- viii) Catheterise → Gross hematuria
- ix) Loss of station (Most characteristic feature of Rupture Uterus)
- x) ⇒ Emergency Laprotomy (Tries to Repair)

\* Can we do Induction in Labour (IOL) in previous C.S

↳ Not C/I

↓

Spontaneous Labour > IOL

↓

DOC ⇒ Oxytocin

Not be given ⇒ Misoprostol

\* Can we do Augmentation of Labour

↳ Not C/I

↳ Continuous fetal Monitoring (+ to identify 1st sign of impending Rupture)

\* Can we do ECV in Previous C.S.

↳ Yes (Not C/I)  
Relative C/I for ECV

(105)

\* Internal Podalic version (IPV) in Previous C.S.

↳ Absolutely C/I in Previous C.S.

\* Induction of Labour is C/I in → Contracted Pelvis

↓  
It means we can't deliver vaginally.

Classical C.S.

H/o Rupture uterus;

Transverse lie;

Major degree of Placenta previa;

Category 3 FHR tracing

↳ Ominous to baby

↳ Immediate delivery

\* Pre-Induction Score → to Induce Labour  
(Bishop's Score)

|                                               | <u>Score</u> |              |          |        |
|-----------------------------------------------|--------------|--------------|----------|--------|
| <u>Cervix</u>                                 | 0            | 1            | 2        | 3      |
| <u>Position (Leaf)</u><br><small>imp.</small> | Posterior    | Mid-position | Anterior |        |
| <u>Consistency</u>                            | Firm         | Medium       | Soft     |        |
| <u>Effacement</u>                             | 0-30%        | 30-50%       | 60-70%   | > 80%  |
| <u>Dilatation (Most imp.)</u>                 | Closed       | 1-2cm        | 3-4cm    | > 5cm  |
| <u>Baby's station</u>                         | -3           | -2           | -1, 0    | +1, +2 |



In Modified Bishop  $\Rightarrow$  Cx effacement is Replaced by Cx Length

In Simplified Bishop  $\Rightarrow$  dilatation  
effacement  
station

\* All

Scores  $\geq 9$  ( $>8$ )  $\Rightarrow$  Favourable  $\rightarrow$  Initiate Uterine Contraction

$\leq 5$  ( $<6$ )  $\Rightarrow$  Unfavourable

Total Score -13

$\hookrightarrow$  firstly do cervical Ripening

Medical  
Methods

Mechanical  
Methods

- Celviprimegel  
(Dinoprostone)  
PGE<sub>2</sub>

0.5mg

Ideal  $\rightarrow$  Intracervical

Acceptable  $\rightarrow$  Posterior fornix

- Put 6 hrly Max<sup>m</sup> 3 doses  
in 24 hrs

- Mlc used

- gt do only Ripening; so,  
after Ripening; give oxytocin  
+0 uterine contraction (after  
6 hr.)

- Misoprostol  
(PGE<sub>1</sub>)

$\downarrow$   
25  $\mu$ g/hr  
vaginally

- Put every 4 hrly

Max<sup>m</sup> 6 doses/24 hrs.

- Better

(do Ripening + Uterine contraction)

- after 4 hrs we give oxytocin; if  
uterine contraction is Not

- Laminaria Tents  
(osmotic dilators)

- Bulb of Foley catheters

## TWINS PREGNANCY

209

106

\* M/c type  $\Rightarrow$  Dizygotic (70+); k/as "Fraternal twins"  
Monozygotic (30+); k/as "Identical twins"

\* In ART procedure  $\Rightarrow$  M/c type  $\Rightarrow$  Dizygotic twins

\* Incidence of Dizygotic twins

Varies

depend on  $\Rightarrow$  Race  
ethnicity  
Family History  
ART

Incidence of Monozygotic twins

Constant

M/c type of Monozygotic twins

$\hookrightarrow$  MCDA

\* Dizygotic  $\Rightarrow$  Dichorionic  
Diamniotic

M/c type  $\longrightarrow$

- depend on time of cell division

Clin 72 hrs of Fertilization  $\Rightarrow$  MCDA

Clin 4th-8th day "  $\Rightarrow$  MCDA

Clin 8-12th day "  $\Rightarrow$  MCMA

Clin >12th day "  $\Rightarrow$  Conjoined twins  
(Siamese)

\* Monozygotic twins has higher Risk as compared to Dichorionic

\* Marker of Dichorionicity (to know chorionicity we do USG in 1st trimester (>7wk))

i) 2 separate Placenta on USG

ii) Opposite sex twins;

iii) Twin Peak sign (Lambda sign)  
(Seen 10-14 week)

$\hookrightarrow$  b/c chorion enters into  
diagonal position

\* In Monochorionic twins

↳ Inverted "T" sign seen on US

iv) 4 Layers in dividing Membrane  
2 Amnion 2 chorion

v) Thickness of dividing Membrane

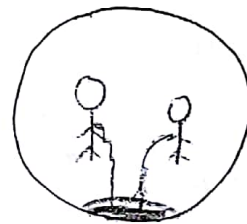


$\geq 2\text{mm}$  thick  $\Rightarrow$  Dichorionic

if  $< 2\text{mm}$  thick  $\Rightarrow$  Monochorionic

\* Monochorionic Monoamniotic twins

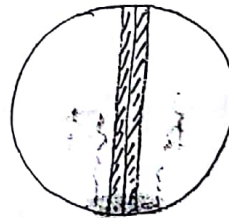
↳



\* M/c type of conjoined twins  $\Rightarrow$  Thoracopagus  
↳ to joint

\* Monochorionic Diamniotic twins

↳



\* M/c Risk to fetus in twin ♀  $\Rightarrow$

also  $\Rightarrow$

Preterm Labour

GCA  $\uparrow$

GUGR

} comparison to  
Single-ton ♀

\* 2 things not seen in Multiletal @

↳ Post dated @

Macrosomia

(107)

\* Fetal Reduction :⇒

- Ideal time to do ⇒ 10-13 week
- Under USG guided Inject KCl into thorax
- Converted into 2 betw (Not less than twins)

\* Monochorionic twin (specific) complication ⇒

- TTTS
- TRAP (Twin Reversed Arterial Perfusion)
  - ↳ klas "Acadiac twinning"
- TAPS (twin Anemia Polycythemia Sequence)
- Selective GUGR
- TT Risk of GCA

TTTS (Twin twin Transfusion Syndrome)

- It is seen in Monochorionic Diamniotic

↳ Reason for TTTS

↳ Vascular Anastomosis

Deep Artery of one baby anastomoses  
with Deep vein of other baby

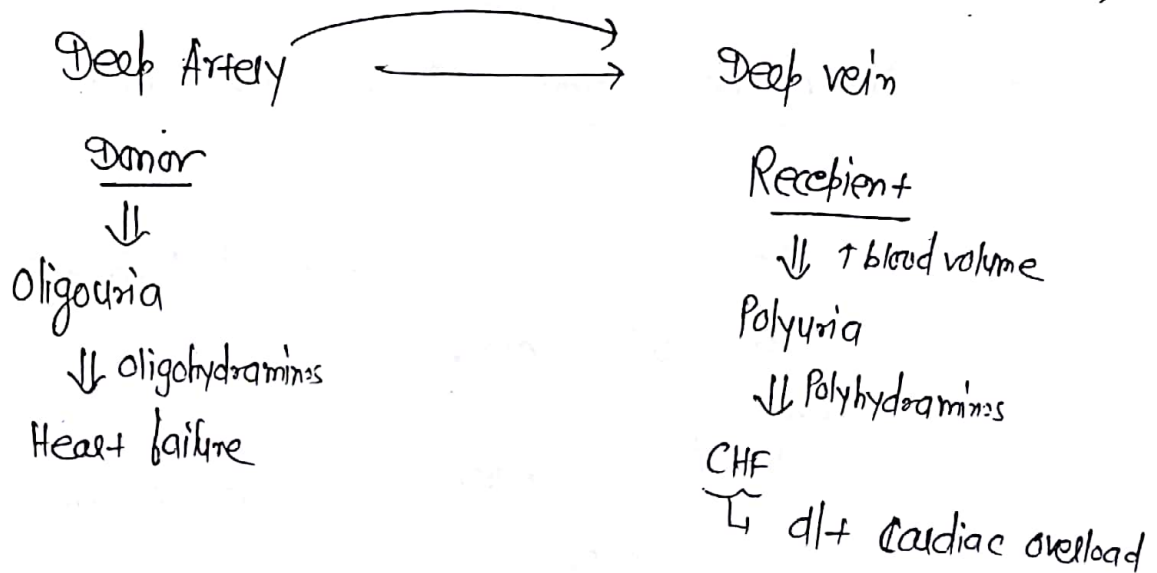


\* In Monochorionic Monoamniotic twins

↳ Plenty of Superficial Artery-Artery  
Anastomosis ⊕; so; TTTS  
not seen in it

Diagnosis ⇒ USG

Simultaneously — Polyhydramnios (by DUP Method  
— oligohydramnios (we see Amniotic fluid)



\* QUINTERO STAGING ⇒ Staging of TTTS

Stage 1 → Poly/oligo + Bladder of oligo twin is visible + Doppler (N)

Stage 2 → Poly/oligo + Bladder of oligo twin is Not visible + Doppler (N)

Stage 3 → Poly/oligo + Bladder Not visible + Doppler (AbN)

Stage 4 → Either / both hydrops fetalis

Stage 5 → Either / both GUD.

TIT  $\Rightarrow$  Fetoscopic Laser Ablation of the vascular Anastomosis. (108)

Stuck twin  $\Rightarrow$  <sup>alive; only stuck</sup> Oligo twin of twin twin transfusion sx.  
 $\downarrow$  can't move; so; Looks like stuck

Vanishing Twin  $\Rightarrow$  Spontaneous abortion of one of the twin.

Fetus Pappeyruerus  $\Rightarrow$  One twin dies & is compressed by other twins.

Impending death  
 $\downarrow$   
 do TOP  
 $\downarrow$   
 take out both the babies; as other baby may go to hypoxic injury

if one twin - already die  
 $\downarrow$  L go  
 continues pregnancy  
 + Monitors the other twin  
 $\downarrow$   
 Baby may go hypoxic injury & coagulation defect both

Superfetation  $\Rightarrow$  different cycle (Menstrual)

Superfecundation  $\Rightarrow$  Same cycle

OUTCOME OF TWINS  $\Rightarrow$  Lie of 1<sup>st</sup> Twin

1<sup>st</sup> - Twin - Longitudinal - vaginal delivery

Non-Longitudinal - C.S.

\* M/C combination in Labour  $\Rightarrow$  Vertex - Vertex  
 1<sup>st</sup> twin                      2<sup>nd</sup> twin

Vertex - breech

Q. Q.

$M_x = 22$  i/b 1st twin = Breech; 2nd = vertex

Vaginal delivery is Not c/I

C.S. > Vaginal delivery

Complication  $\Rightarrow$  twin Interlocking

Q. Q.

$M_x = 22$  i/b 1st vertex deliver; 2nd breech

$\Downarrow$

Assisted Breech vaginal delivery

Q. Q.

$M_x = 22$  i/b 1st vertex deliver; 2nd Transverse lie

$\Downarrow$

do Internal Podalic version (IPV)

$\subset$  Breech extraction

gives under  
GA (General Anesthesia);  
Mother have no effect  
to push the baby; so, do  
Breech extraction

$\hookrightarrow$  i.e. No efforts by Mother at all

\* The lie of 2nd baby is Not  
decided until 1st baby is delivered

Q. Q.

M/c complication of Monoamniotic twins are

$\hookrightarrow$

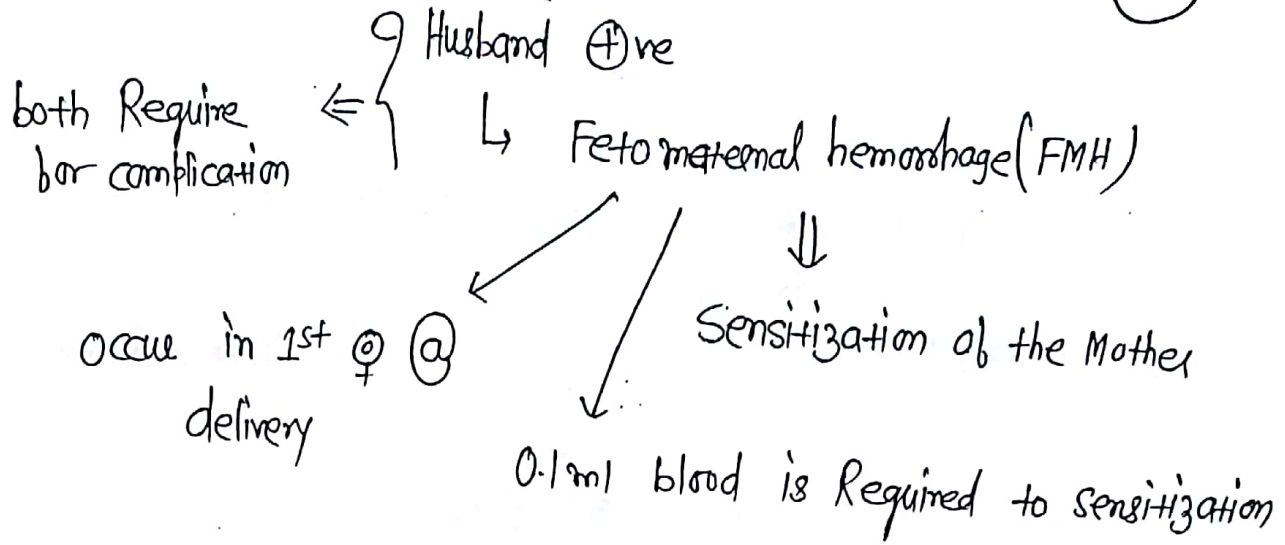
Cord Entanglement

$\hookrightarrow$  delivered by CS:

$\Downarrow$

by 32-34 weeks

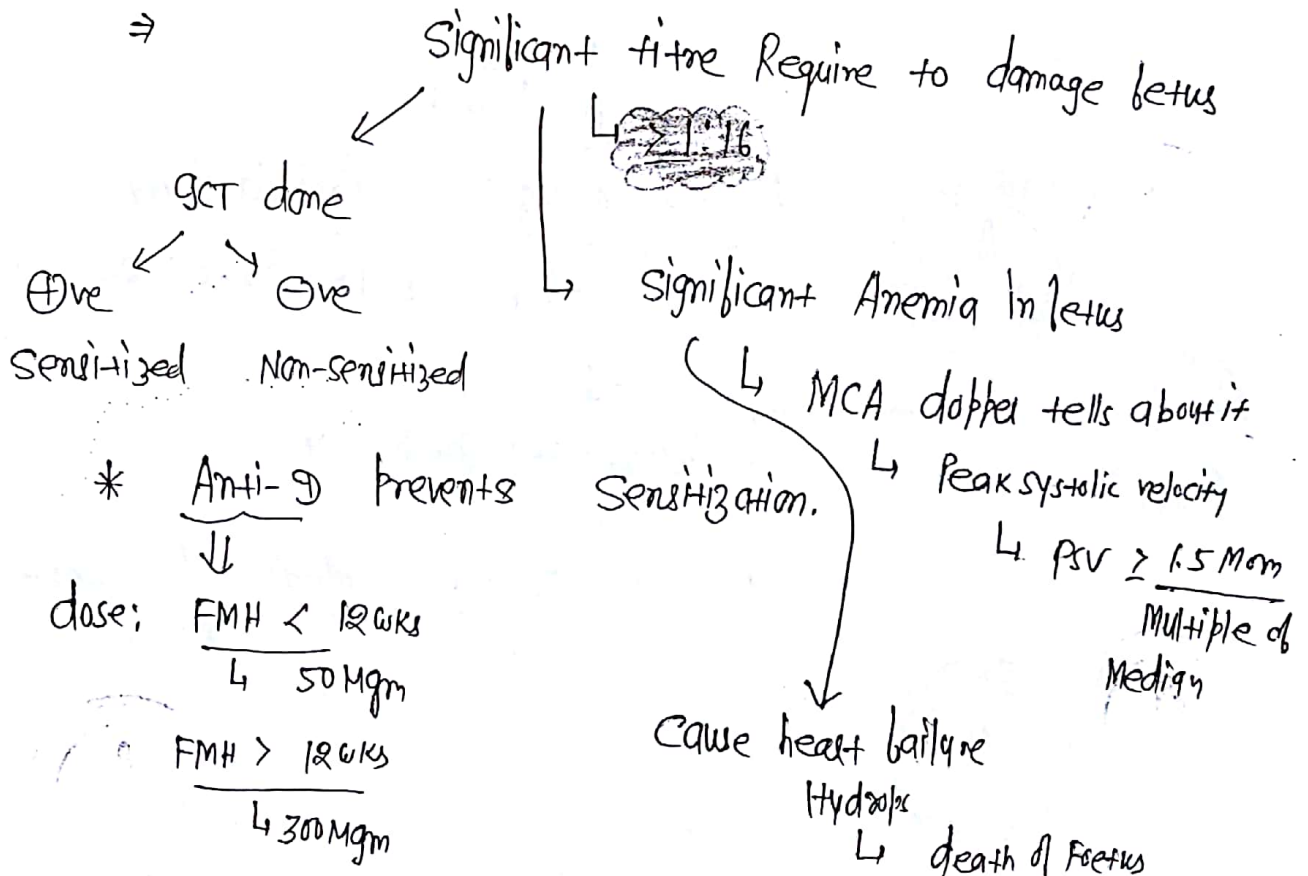
(109)

Rh  $\ominus$ ve PREGNANCY

$\Rightarrow$  1st Ab produced by Mother after sensitization

$\hookrightarrow$  IgM (doesn't cross the placenta & Not cause affect in baby in 1st  $\ominus$ )

Later she produce IgG (cross placenta)





\* 300 Mgm of Anti-D Neutralize [ fetal blood - 30mL  
fetal RBC - 15mL

\* if we suspect FMI more than usual then to calculate dose of Anti-D — Kb test  
(Kleihauer-Betke)

Case I →

Rh ⊖ve  
↓  
Husband ⊕ve  
↓  
@ 12 week GCT ⊖ve  
(Not sensitized)

↓  
GCT (28wk)

⊕ve

↓  
Anti-D 300-Mgm → Prophylactically

↳ this works for 12wks; so she delivers after safely.

↓  
if Baby blood group ⊕ve

↓  
Anti-D 300 Mgm (ideally 72hrs; can be given up to 28 days)  
i/m.

Case 2 →

Rh +ve

(110)



Husband Rh +ve



① 12 weeks

GCT +ve  
( $< 1:16$ )

(Anti-D has No Role in GCT +ve Patient; it Means Sensitization already takes place)



Repeat GCT x 4 weekly

if Rising trend; Repeat 2 weekly



deliver at term

Case 3 →

Rh +ve



Husband Rh +ve

② 28 weeks GCT +ve  
( $> 1:16$ )if Hb level  $< 5 \text{ gmt}$  of fetus

Hydrops fetalis

↓ followed up for severe Anemia

PSV - MCA doppler

 $\geq 1.5 \text{ Mom}$  $< 1.5 \text{ Mom}$ 

↳ Repeat MCA doppler

P.O.G.  $\geq 34 \text{ wk}$ ↓  
deliverP.O.G.  $< 34 \text{ weeks}$ 

↓ do cordocentesis

Intrauterine transfusion

Hb  $< 8 \text{ gmt}$

Hydrops fetalis  $\Rightarrow$  Most common cause (Non-immune Mediated)

$\downarrow$

M/c/c

$\downarrow$  Immune Mediated

$\downarrow$  CVS - abnormalities

Infection that can cause hydrops fetalis  $\Rightarrow$  Parvovirus B-19

USG diagnosis  $\Rightarrow$  Any  $\geq 2$  of the following

- i) Pleural effusion
  - ii) Pericardial effusion
  - iii) Ascites
  - iv) Subcutaneous edema
- } Criteria

Scalp edema  $\Rightarrow$  Buddha sign.

Findings in hydrops fetalis  $\Rightarrow$  Placental Megaly  
Polyhydramnios

\* Gene for Rh factor Located on  $\Rightarrow$  Short Arm of chr-1

# HEART DISEASE IN PREGNANCY

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(11)

## Symptoms

- Orthopnea
- Paroxysmal Nocturnal dyspnea
- exertional dyspnea (Progressive)



It is Physiological; but progressive exertional dyspnea is Pathological

## Signs -

- Cyanosis / clubbing
- Diastolic Murmur
- Systolic Murmur > grade 2
- Cardiomegaly
- Atrial fibrillation / ABN Rhythm
- CHF
- Persistently distended Neck veins
- Pulmonary Artery HTN  
↳ Loud P<sub>2</sub>
- wide split S<sub>2</sub>

\* M/c heart disease in ♀ ⇒ Mitral Stenosis

M/c congenital heart disease in ♀ ⇒ ASD

M/c congenital valvular heart disease ⇒ Mitral valve Prolapse

\* High Mortality Rate (> 50%) in ♀ ⇒

- i) Marfan's sx ⇔ Aortic Root Involvement
- ii) Coarctation of Aorta ⇔ Aortic valve Involvement
- iii) Eisenmenger Syndrome

→ TOP (Abortion)

In Severe MS ( $< 1.5 \text{ cm}^2$ ) <sup>valve area</sup>



v) NYHA grade 3/4

vi) Ejection fraction  $< 45\%$

\* good outcome



ASD

Corrected TOF

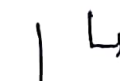
VSD

Mitral valve Prolapse

PDA

Ebstein Anomaly

\* if a patient has severe MS



ideally goes to - Valve Replacement

High Mortality  
Rate  $\Rightarrow 30\%$

Sx preconceptually



Not be done during  $\varnothing$

↳ if she wants to continue  $\varnothing$

↳ do Balloon valvotomy

↳ ideal time

↳ 18-20 weeks

\* if she has valve Replacement



Prosthetic heart valve  $\rightarrow$  Pt. is on Anti-coagulation

↓ if she wants to conceive

Antidote for heparin  
↓  
Protamine Sulfate  
Warfarin has No Antidote

1-12 weeks  $\rightarrow$  Heparin

12-36 weeks  $\rightarrow$  Warfarin  
(More potent)

36 - onset  $\rightarrow$  Heparin

causes embryo pathy in  
1st trimester



Chondrodysplasia Punctata  
(Stippled femoral epiphysis)

On close to Labour;

if warfarin given; PPH happen also

\* During Labour → Stop Anticoagulant

221

(112)

However Restart the Anticoagulant In vaginal delivery ⇒ Alter 6hr  
In CS ⇒ Alter 24hr

⇓

Restart = Heparin + Warfarin

⇓

With draw Heparin ; core  
- d INR ; As Warfarin has  
delayed onset of Action

### \* GENERAL PRINCIPLES :-

i) Max<sup>m</sup> Rise of CHF :-

Immediate Post partum > 2nd stage > 32 weeks

ii) vaginal delivery is preferred;

C.S. is Reserved for obstetric Indication

iii) Heart disease Indication of C.S. ⇒

i) Marfan's Sy = Aortic Root Involvement

ii) Coarctation of aorta = Aortic valve Involvement

iii) Aortic dissection

iv) Severe AS

In all Ejection fraction is affected

\* In Eisenmenger's Sy ⇒ vaginal delivery preferred

\* Induction of Labour (IOL) is not contraindicated in heart disease

↳ IOL is safe

however; Spontaneous Labour over IOL

QA 38 wks - Heart failure; K/d/o Heart disease;

↓  
Mx ⇒ Stabilize the patient

↓  
Wait for Spontaneous Labour

i) Propped up / Left Lateral Posture / O<sub>2</sub> by Mask - Readily available

ii) Restrict iv fluids @ 75 mL/hr

A.S. ⇒ wet side

iii) Restrict the phv exams

iv) ARM — Can be done

Memb. Rupture — Prophylactic Antibiotics  
(AHA guidelines) ↓  
Ampicillin + Gentamicin

↓  
Vaginal delivery safe

v) Pain Management ⇒ Epidural Analgesia  
(Neuraxial)

ix) Cut Short the 2nd Stage of Labour



Forceps delivery > Vacuum delivery

(113)

x) Immediately after delivery

- Given Gn. Lasix (to ↓ Preload)
- Avoid Methargin

Hang down the patient leg from delivery table (to ↑ Venous Return)

xi) C.S. ⇒ Anesthesia

→ SLE ⇒ Hypotension

Severe A.R. }  
Severe A.S. } ⇒ Low Ejection fraction  
Cyanotic H.D. }  
\* In Aortic dissection (emergency cond<sup>n</sup>) do emergency C.S. under G.A.

### PERIPARTUM CARDIOMYOPATHY

- Development of heart failure Around Labour in a woman w<sup>o</sup> No underlying Heart Disease
  - ↳ 1 Month before delivery to 5 Month after delivery
- Ejection fraction - Low
- Left ventricle May or Mayn't be dilated
- Mainly in Pre-eclampsia patient; also in Multi-fetal @; advanced Maternal age
- Prolactin has some role to develop
- Mx = Same as Heart failure



# Fetal Monitoring

Fetal Movements  $\Rightarrow$  Quickening (1st Fetal Movement)

$\hookrightarrow$  16 weeks — Primigravida  
18 weeks — Multigravida

(N)  $\geq 10$  fetal Movement in a 2 hr period of Rest  
OR  
 $\geq 10$  Fetal Movement in a 12 hr period in Routine activity.

Max<sup>m</sup> fetal Movement  $\Rightarrow$  @ 32 weeks Perceived by women

— earliest time for gud in Absence Fetal Movement = 12 hrs  
Max<sup>m</sup> time for gud in Absence Fetal Movement = 48 hrs

— Modified Biophysical Profile (BPP)  $\Rightarrow$  ↓ Fetal Movement beyond 32 weeks

also klas "cardio-tocography"

(NST)

$\Downarrow$   
In Acute Injury

$\gg$

(AF)

$\Rightarrow$  Amniotic fluid

$\hookrightarrow$  In Chronic Injury (UPI)

(N) Heart Rate = 110-160 beats/min.

Beat to beat variability = 5-25 beats/min

Acceleration  $\Rightarrow$   $\uparrow$  Heart Rate by 15 beats/min above baseline for 15 sec

(114)

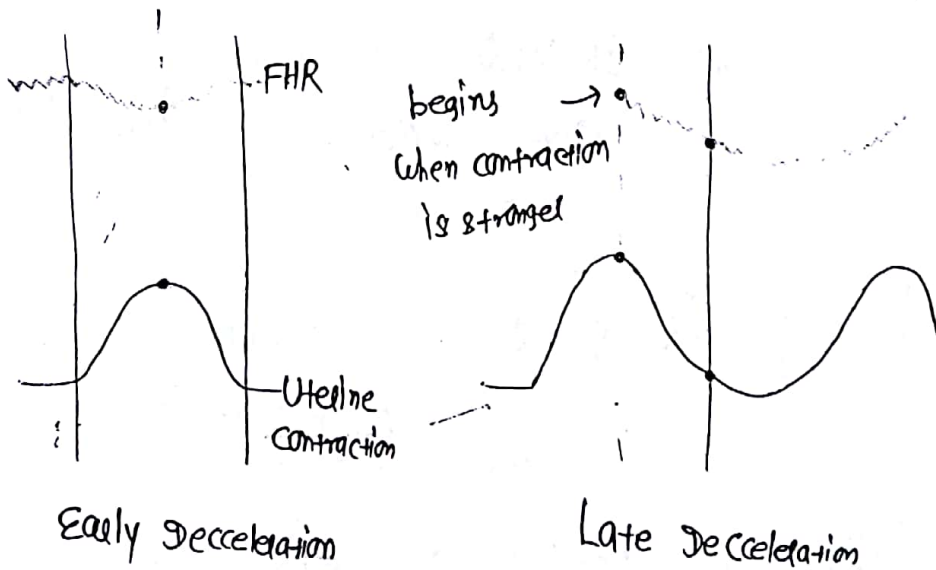
Deceleration

(3 types)

gradual  
dip in heart  
Rateit is  
also bad  
outcomeEarly  $\Rightarrow$  dip in heart Rate (d/t head compression)Late  $\Rightarrow$  dip persisting after the uterine contraction ends (d/t uteroplacental insufficiency)Variable  $\Rightarrow$  Abrupt dip in heart Rate (d/t cord compression)Sinusoidal  $\Rightarrow$  Marker of Severe fetal Anemia  
Severe fetal hypoxia

$\hookrightarrow$  Most ominous type of Pattern  
(worst outcome)

When variable deceleration is significant

 $\hookrightarrow$  i) if  $HR < 70 \text{ beats/min} \times 1 \text{ min}$ ii) Persistent Around  $\geq 50\%$  of uterine contraction

Reactive NST  $\Rightarrow$  two or more than two ~~de~~ acceleration  
in a span of 20 min.

Non-Reactive NST  $\Rightarrow$   $< 2$  acceleration in a span of  
40 mins  
 $\hookrightarrow$  Physiologically if baby is sleeping

\* Category 1

Beat-  
Beat+  
variability

(N)

Category 2

$\downarrow$

- Further assessment
- LLP (Left Lateral Posture)
- O<sub>2</sub> by Mask
- Stop oxytocin
- IV fluids
- Give tocolytics

Immediate delivery + CS.  $\leftarrow$  ominous

Category 3

Absent  $\bar{c}$

Any one of the following

$\downarrow$

Bradycardia

Late deceleration

Persistent variable deceleration

Sinusoidal

NST in high Risk  $\phi$   $\Rightarrow$  twice a week  
(once in 72 hrs)

\* BPP (Biophysical Profile)  $\Rightarrow$  Manning Score  
 $\hookrightarrow$  Report card of Fetus

→ USG for 30 min

(15)

↳ use a score of +2 or zero

- It has 5 components

↓

Breathing Movement ⇒ At least one Movement Lasting 30 sec +2

Gross Body Movement ⇒ 3 Movements +2

Tone ⇒ Flexion - Extension - Flexion +2

Amniotic fluid ⇒ At least 1 Pocket of 2 cm +2

NST ⇒ Reactive +2

if Score is  $\frac{8,10}{10} \Rightarrow \textcircled{N}$

$\frac{6}{10} \Rightarrow \textcircled{N}$  Liquor = Equivocal = Repeat testing on the same day

$\leq \frac{4}{10} =$  Immediate delivery

\*  $\textcircled{AbN}$  In Acute hypoxic

↓

Loss of Acceleration → Breathing → Gross body → Tone  
↓  
1st to become  $\textcircled{AbN}$  Movement Movement



\* How frequently to be done in high Risk @  
L once in week

**DOPPLER**  $\Rightarrow$  • M/C 1st vessel  $\Rightarrow$  Uterine A  
L signs of UPI

• S/D Ratio

L (N) @ S/D  $\downarrow$ ing

• UPI  $\Rightarrow$  S/D  $\geq 3$

$\downarrow$

$> 28-30$  wk

$\downarrow$

$\geq 3$

UPI

REDF  $\rightarrow$  TOP

• AEDF - TOP  $\geq 34$  weeks

L (a) 34 weeks - gives steroid + further  
or less than 34 weeks Monitoring.

\* **MCA Doppler**  $\Rightarrow$  Not best for UPI

L (N); b/c in UPI; foetus sends blood to vital organs  
 $\downarrow$

Early stage MCA doppler  $\Rightarrow$  (N)

$\downarrow$

Brain sparing effect

\* REDF — Umbilical Artery

↳ Steroids — 2 days

↓  
Reversal in venous Doppler <sup>Last vessel to show REDF</sup>

⇓  
Indicates impending death.

\* MCA Doppler is best studied for fetal Anemia

\* Best for Fetal Monitoring

↳ Fetal Scalp blood pH

(N) ⇒ 7.25 — 7.35 <sup>Repeat test after 30 min</sup>  
7.20 = 7.25 = Borderline

< 7.20 = Acidosis

↳ Immediate delivery

\* VAST (vibroacoustic stimulation test) ⇒

High Intensity Sound waves

Released from Artificial larynx → on Maternal Abdomen  
for 1 sec (to 2 sec)

(N) Response ⇒ ↑ from baseline by 15 bpm in 15 sec  
of stimulus

\* M/c Method for Intrapartum Monitoring →

Intermittent - Heart Rate Auscultation.

1st stage

2nd stage

In Low Risk ♀ - every 30min

every 15min

In High Risk ♀ - every 15min

- every 5min

\* Time to Heart Rate listen ⇒ Immediately after contraction  
& listen for 1 minutes  
(Not 15 sec x 4)







# GYNAECOLOGY $\Rightarrow$ Base of Gynae

- 233

$\hookrightarrow$  Hormones

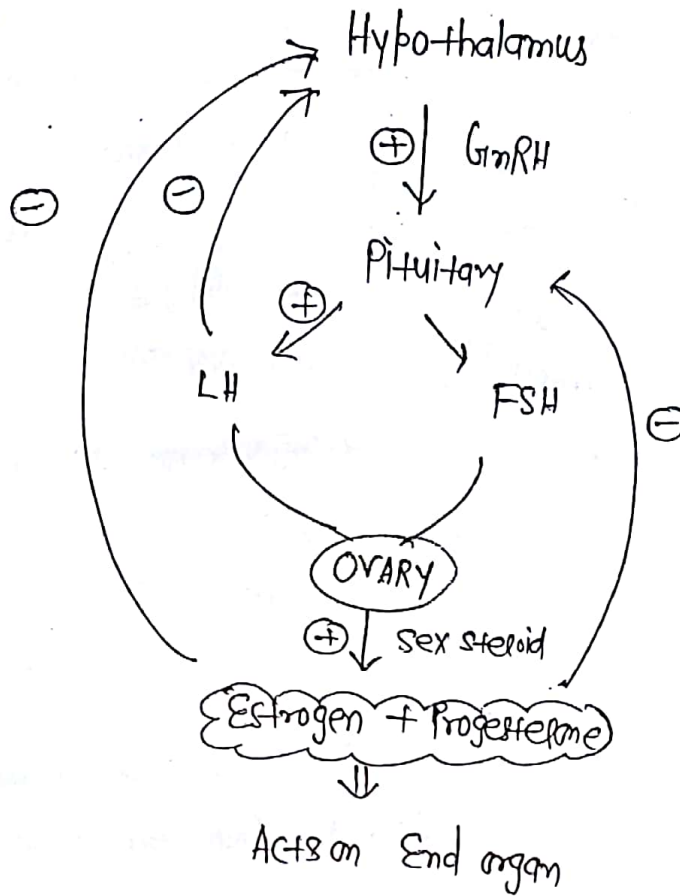
(118)

## \* Hypothalamic-Pituitary-Ovarian Axis (HPO axis) $\hookrightarrow$

$\hookrightarrow$  Not develop before puberty.

Sensitive Around 8-12yr

Fully established by 13-14yr



Feed forward  
Loops

\* In obese  $\phi$  / Pubertal change occur early; diff. level of Leptin

$\hookrightarrow$  Hormone Made by Adipose cells that inhibit hunger.

## \* Estrogen

\*  $C_{18}$  Steroids

Types  $\Rightarrow$   $E_1$  - Estrone - Predominant Estrogen in Post Menopausal  
 $E_2$  - Estradiol  $\hookrightarrow$  in Reproductive Life  
 $E_3$  - Estriol  $\hookrightarrow$  in Pregnancy

Most Potent  $\Rightarrow$   $E_2 > E_1 > E_3$   
 (Natural)

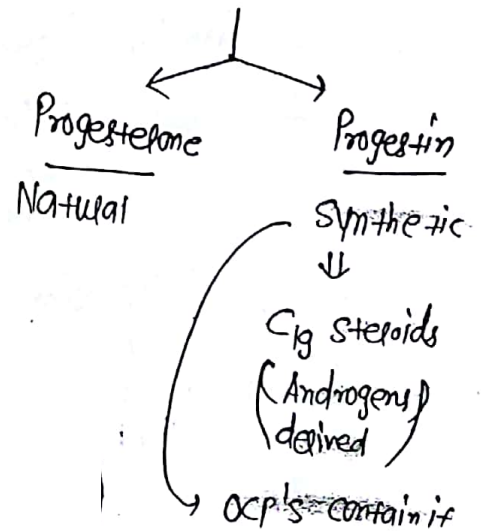
\* Most Potent (Synthetic)

$\hookrightarrow$  Ethinyl-Estradiol (EE)

$\hookrightarrow$  Used in Combined ocp's

## Progesterone

$C_{21}$  Steroids



\* Classification of ocp's on the basis of Amount of Ethinyl

Estradiol  $\Rightarrow$  .

High dose  $\geq 50$  Mgm

Low dose 30-35 Mgm

Very low dose  $\leq 20$  Mgm.

Lowest dose 10 Mgm  
(LoLoestrin)

\* Classification of ocp's on the basis of Synthetic Progestin  $\Rightarrow$

1st generation  $\Rightarrow$  Norethindrone

2nd generation  $\Rightarrow$  Levonorgestrel (LNG)

3rd generation  $\Rightarrow$  Desogestrel (M/C wed)

Gestodene  
Norgestimate.

Used in  
Malan, Mala D

Used in  
Novelon Pills

Anti Androgenic

M/C ocp pills

4th generation  $\Rightarrow$  Anti Androgenic

Spirolactone derivative  
(Drospirenone)

Dienogest

as generation Test Lipid profile side effect  
less & Androgenic side effect also less

\* Sources

$E_1 \Rightarrow$  Post Menopausal

$\Downarrow$   
Peripheral conversion

Androstenedione Adipose tissue  $\rightarrow E_1$   
(Aromatase)

$E_2 \Rightarrow$  Reproductive Age

$\Downarrow$   
comes from granulosa cells  
of ovary  $\Downarrow$  dependent on  
Theca cells

• corpus luteum

2 cell 2 gonadotropin  
Theory.

\* Sources

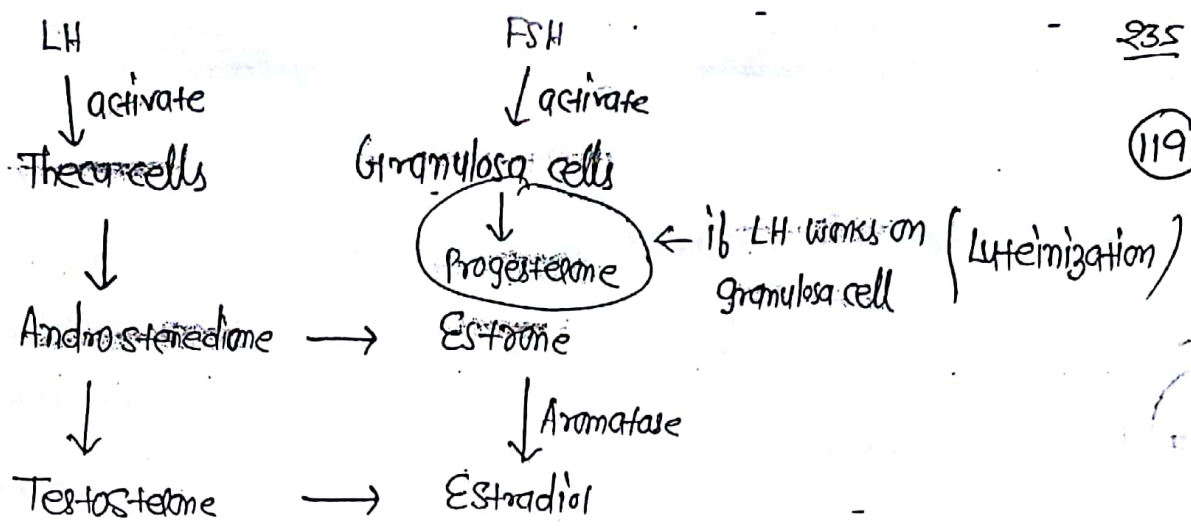
• Ovary

$\hookrightarrow$  Corpus luteum

$\Downarrow$   
Formed by Luteinization of  
Granulosa cells.

• In  $\text{♀}$   $\Rightarrow$  Produced by Placenta

$\Downarrow$   
Precursor (Maternal LDL  
Cholesterol)



\* Theca cell don't have Aromatase enzyme.

\* Granulosa cells don't have 17- $\alpha$ -HSD enzyme ; so can't make Androgen

\* Placenta —  $E_3$  (Fetal DHEA)  
(Pregnancy)  $\rightarrow E_2$

\* Most of estrogen  $\rightarrow$  is in bound form (99%)

1% in free form

\* Bound; 2% free

$\hookrightarrow$  Mainly Albumin

(cortisol binding  $\leftarrow$  CBG globulin) Not with SHBG

Mainly bound  $\bar{c}$

SHBG

also  $\bar{c}$  Albumin  $\rightarrow$  Sex hormone binding  
SHBG (Liver) Globulin  
synthesis

Estrogen

$\rightarrow$

End product

$\hookrightarrow$  Pregnenediol

End products  $\Rightarrow$  Glucuronides  
(sulphonides)

\* Receptor & Location

$\hookrightarrow$  Intracellular

Genoprotective



Uterus

Non-Pregnant

(E)

Endometrial Proliferation

(P)

Protective

Stops Proliferation

↳ do secretory changes

↓  
decidualisation

Growth of uterus

Growth of Uterus

↓

Relaxation of Uterine

Smooth Muscle

Cervical  
Mucous ⇒

Thin  
Copious (Large Amount)  
Watery

Thick  
Scanty

Elastic can be stretched  
by fingers

Spinnbarkeit (↑ X)

No Spinnbarkeit

highly viscous

high amounts of  
NaCl Required to  
secrete  
estrogen to secrete

Ferning → Fern like pattern  
on microscopic exam  
↳ earliest @ 8<sup>th</sup> day  
disappears by 12<sup>th</sup> day  
↳ like "Arborization".

(X) Tack phenomenon  
(X) (No ferning)

Cx Mucous breaks  
on stretching

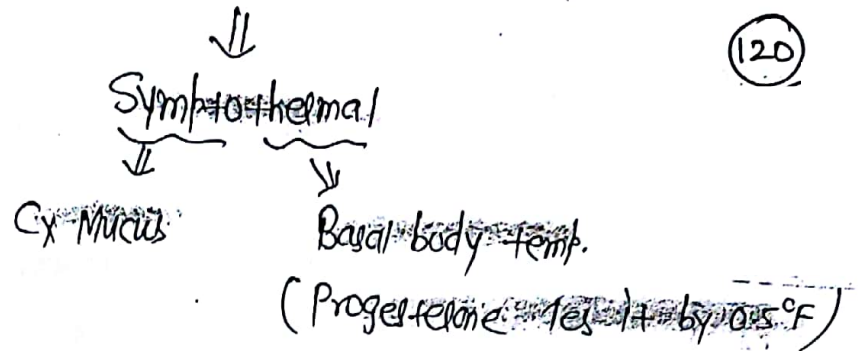
\* Periovulatory Cx Mucous is (E) type ⇒ highly fertile.  
↳ test of ovulation

\* Mechanism of POP's (mini pills)

↳ to make the Cx Mucous thick  
(Alteration of Cx Mucous)

\* Cx Mucous ⇒ Natural Method of contraception  
↳ "Billings Method of Contraception"

Q8 Which Natural Method is best (least failure rate)



→ Cx Mucus is also impermeable to Micro-organism

(P) + type

$\Downarrow$   
Natural defense Mechanism

→ Mirena will ves the Risk of Pelvic Inflammatory disease

|                | (E)                                                    | (P)                                                                 |
|----------------|--------------------------------------------------------|---------------------------------------------------------------------|
| Fallopian Tube | <p>↑ Motility of Fallopian tube</p> <p>↓ secretion</p> | <p>↑ Secretion</p> <p>↓ Motility</p> <p>POP → R/F for ectopic P</p> |

Vaginal Cytology ⇒

→ Big cell Nuclei become small; Pyknotic & lades

Superficial cells → Pink in colour

Cytoplasm may contain the vacuoles

High karyopyknotic Index (Small Nucleus)

Intermediate cells

In Post-Menopausal ⇒ Parabasal/Basal cells

↳ Small cell, Big Nucleus

tells about Hormonal Status Parabasal cell : Intermediate cell : superficial cell

Maturation Index

0 : 10 : 30 : 40 : 50 : 60 : 70 : 80 : 90 : 100 ⇒ ♀

0 : 10 : 30 : 40 : 50 : 60 : 70 : 80 : 90 : 100 ⇒ Periovulatory ♀

100 : 0 : 0 : 0 : 0 : 0 : 0 : 0 : 0 : 0 ⇒ Post Menopausal Post partum.

(E)

(P)

Effect on  
Salt & water

Retention

Excretion

Cholesterol

↑ HDL

↓ HDL

↓ LDL

↑ LDL

↑ Triglyceride

(Total cholesterol ↓)

↓

Cardioprotective

Bones

Causes Mineralization  
of bone

No effect on bone

- epiphyseal closure

[ Post Menopausal → Osteoporosis  
[ Precocious Puberty + ↓ growth ]

S. Ca<sup>2+</sup> Level

↓

Urinary excretion

↓



Coagulation  
Profile

(E) Hypercoagulable state <sup>++</sup> Inhibits Fibrinolysis  
2, 7, 8, 10

239

(P)

No effect,  
so, can be  
well

H/o Venous Thromboembolism  
Stroke  
CAD  
all absolute  
C/I of ocp.

(121)

Estrogen → Causes upregulation of Progesterone Receptors on the Endometrium

Progesterone → Down Regulation of Estrogen Receptors on the Endometrium

Progesterone acts only on Estrogen primed Endometrium

Estrogen affects to higher centre ⇒

(E)  $\xrightarrow{\ominus ve}$  FSH

(E)  $\xrightarrow[\ominus ve]{\text{In Low Amount}}$  LH

$\xrightarrow[\oplus ve]{\text{In High Amount}}$  LH

Neuroendocrine phenomenon

High Amount  
of Estrogen

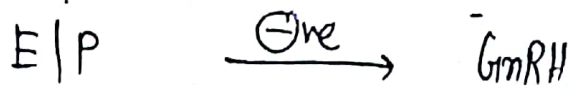
Initiation

LH Surge

(P) Low Amount  $\xrightarrow{\oplus ve}$  LH/FSH

High Amount  $\xrightarrow{\ominus ve}$  LH/FSH





## GONADOTROPINS

- Released by Anterior pituitary;
- Basophilic cells secrete
- Pulsatile
- Protein hormone



FSH  $t_{1/2} = 3-4 \text{ hr}$

LH  $t_{1/2} = 20 \text{ min}$

FSH  $\textcircled{R}$  Gn females  $\rightarrow$  Granulosa cells

Gn Males  $\rightarrow$  Sertoli cells (Spermatogenesis)

LH  $\textcircled{R}$  Gn Females - Theca cells

Granulosa cells - appear only in Late Proliferative Phase

Gn Males - Leydig cells

$\hookrightarrow$  Produce Testosterone

FSH  $\Rightarrow$  i) do Selection of cohort of follicle every month

(OOO)  
(OOO)

ii) Selection & growth of dominant follicle.

ii) Ovulation → Final Release of Ovum by collagen breakdown is brought about by FSH

(122)

LH → Function

- i) Ovulation
- ii) Formation & Maintenance of Corpus Luteum
- iii) Final Growth of Follicle

LH Surge  $\xrightarrow[24-36 \text{ hrs}]{36 \text{ hrs}}$  Ovulation

LH Peak  $\xrightarrow{12 \text{ hrs}}$  Ovulation

\* LH Surge ⇒ Initiation by high level of Estrogen.  
 (200 pg x 48 hrs)  
 ↳ Amount of estrogen to cause LH surge

Maintenance of LH Surge ⇒ E + P

\* When does Progesterone Synthesis begin  
 ↳ Before ovulation (36 hrs) → Low in Amount

LH → Surge → Luteinization of Granulosa cells

Q. Just before ovulation; which is true?

- (a) ↑ LH; ↓ FSH
- (b) ↑ FSH; ↓ LH
- (c) Both ↑ (LH Peak >> FSH Peak) → Small amount of Progesterone gives ⊕ feedback.
- (d) Both ↓

CORPUS LUTEUM  $\Rightarrow$  Every Month  $\Rightarrow$  die

$\hookrightarrow$  Life span 14 days (constant luteal phase)

QA if 36 day Menstrual cycle ; ovulation day "

$\Downarrow$   
on 22nd day

QA Which hormones Maintain the corpus Luteum

$\hookrightarrow$  (LH)

QA Which hormones Maintain the corpus Luteum in ♀

$\hookrightarrow$  (HCG)

Rescue the corpus luteum from Luteolysis

Corpus Luteum

$\hookrightarrow$  Progesterone

Estrogen

Relaxin

Inhibin A

Secreted by granulosa  
cell of the follicle

Inhibin B

$\searrow$   
Inhibit the Release of FSH

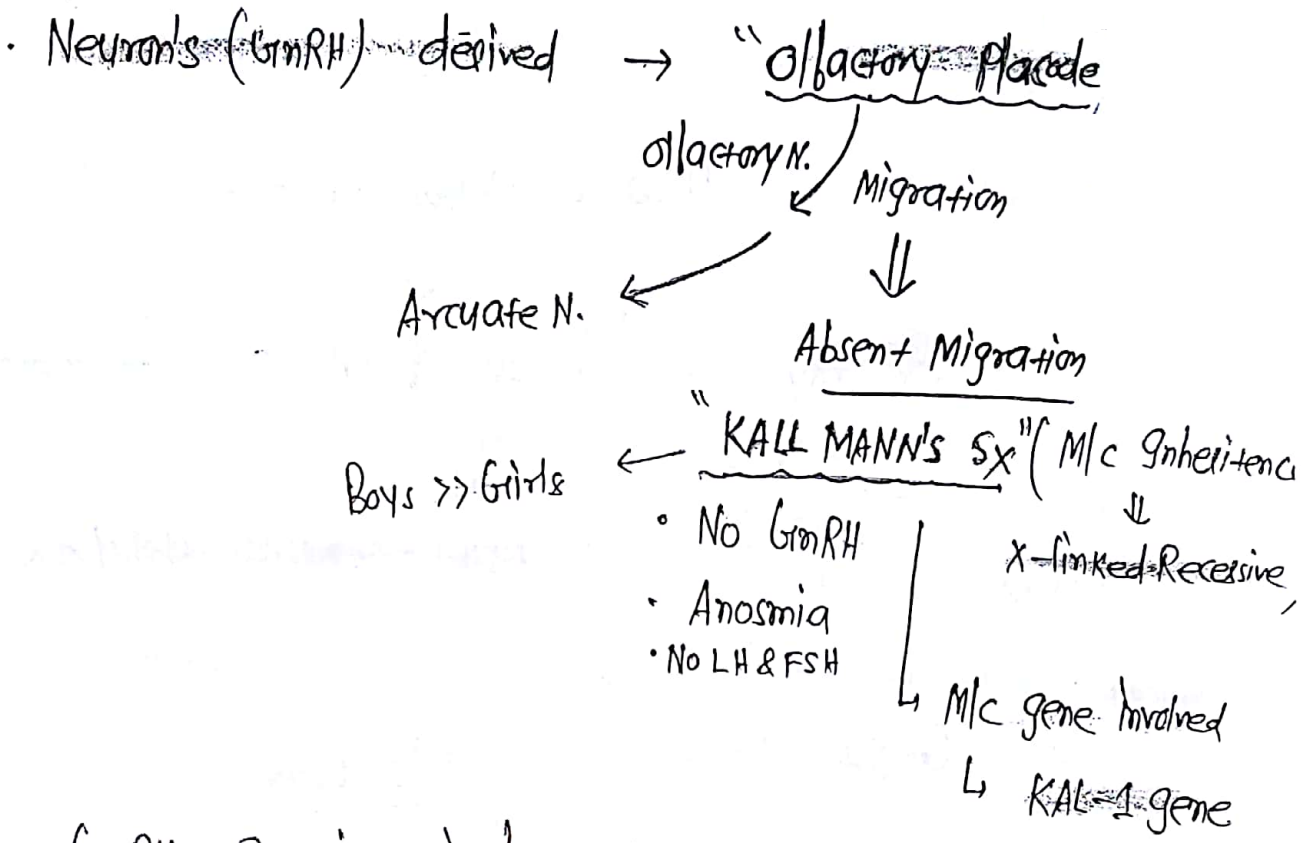
- Peak activity of corpus Luteum  $\Rightarrow$  8th day Post ovulation

$\swarrow$   
Maxim Progesterone production

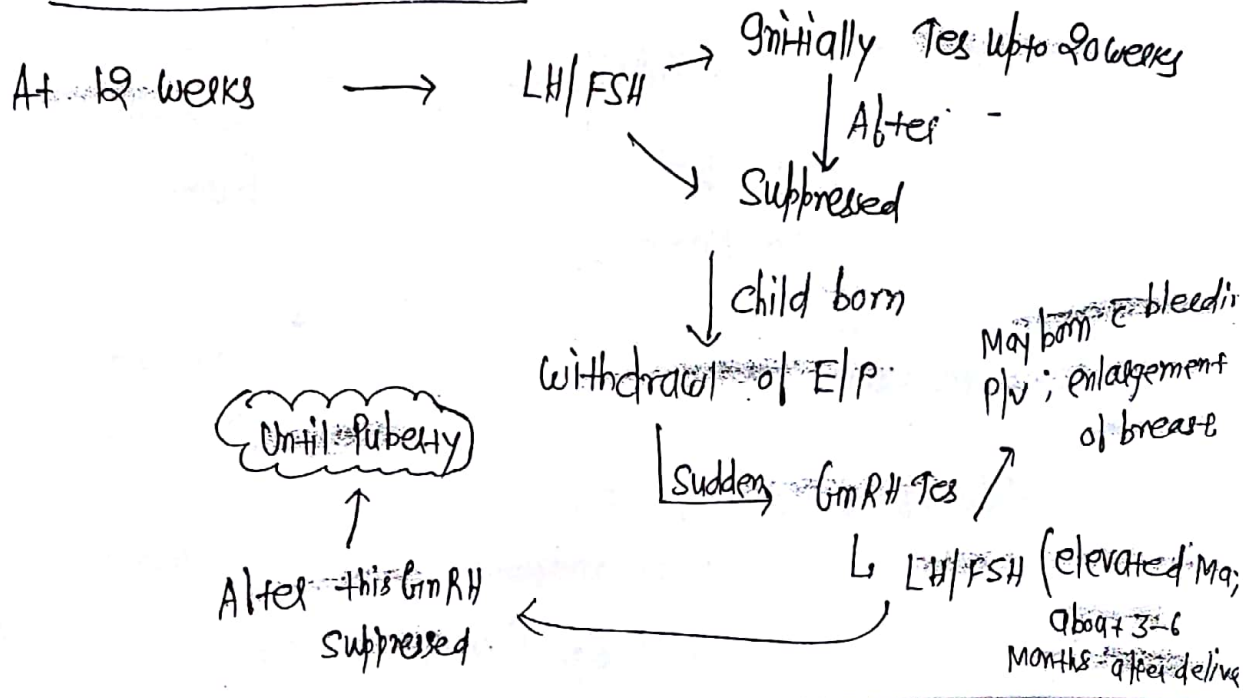


# HYPOTHALAMUS → Release GnRH

GnRH — Arcuate Nucleus Released if (in Medial hypo-thalamus)  
 ↳ decapeptide  
 ↳  $t_{1/2} = 3-4 \text{ min}$

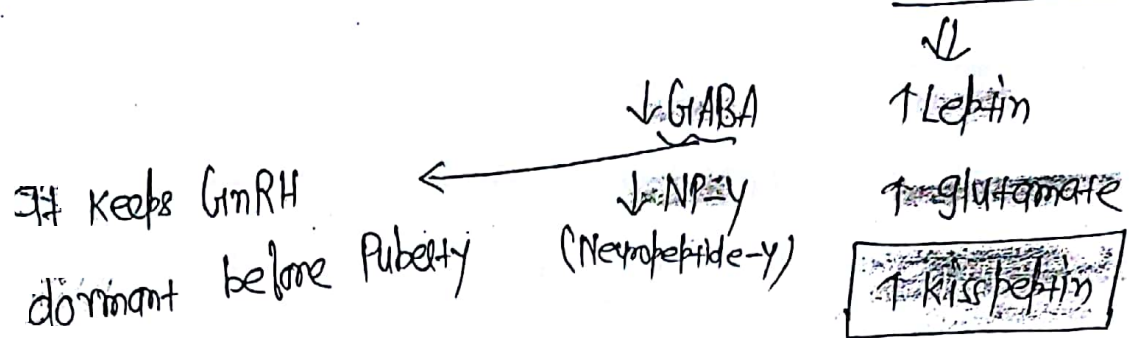


## GnRH Secretion in fetus

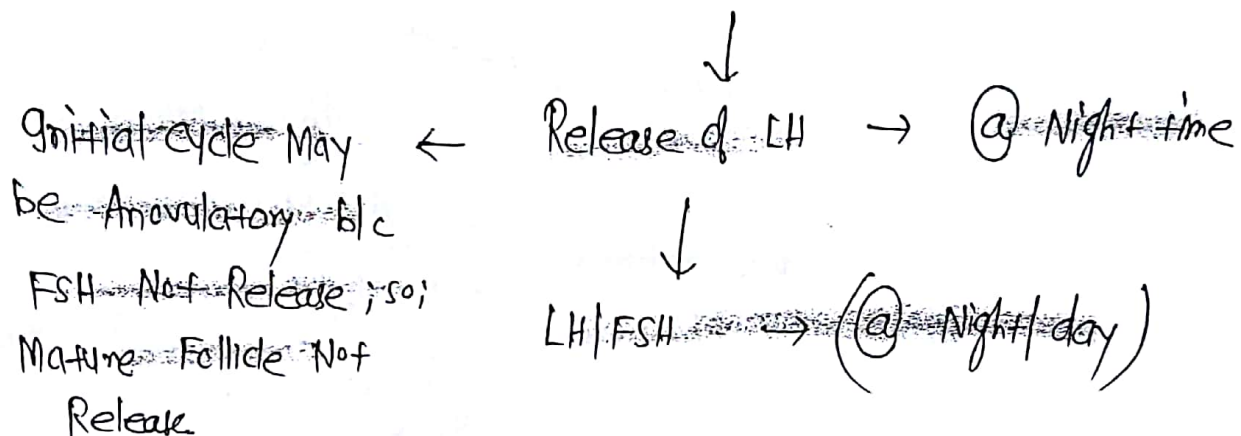




\* Activation of HP axis @ Puberty  $\Rightarrow$  ~~dit Neurotransmitter~~



Pulsatile Release of GnRH  $\rightarrow$  Night time

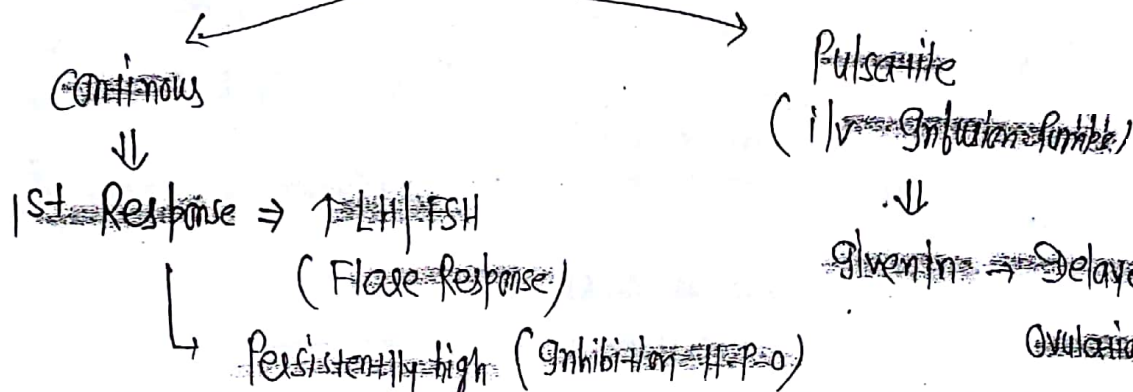


\* GnRH Agonist  $\Rightarrow$  M/c used

$\downarrow$   
 Not effective orally.

give in two Names

L Luprolide  
 (Goserelin  
 Nafarelin  
 Buserelin)



ContinuousPulsatile

(24)

Endometriosis

Kallmann's Syndrome

Fibroid UterusHirsutismPrecocious PubertyBreast CancerProstate Cancer\* GnRH Antagonist → M/c used ⇒ Cetrorelix\*

↳ No Flare Response (No Initial Tes)

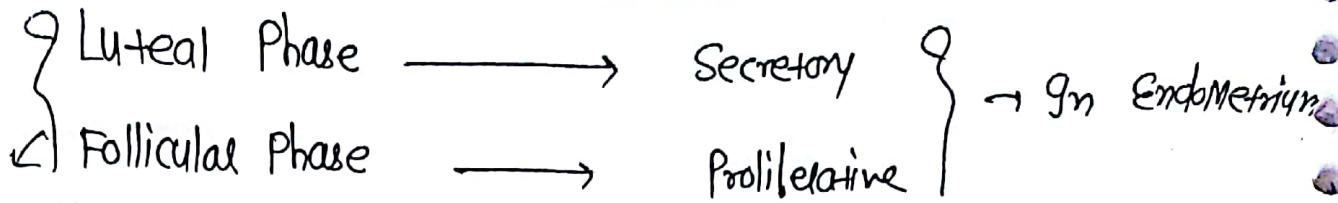
Same Antagonist - orally active

Indication ⇒ Same as Continuous GnRH Agonist Indication

\* GnRH

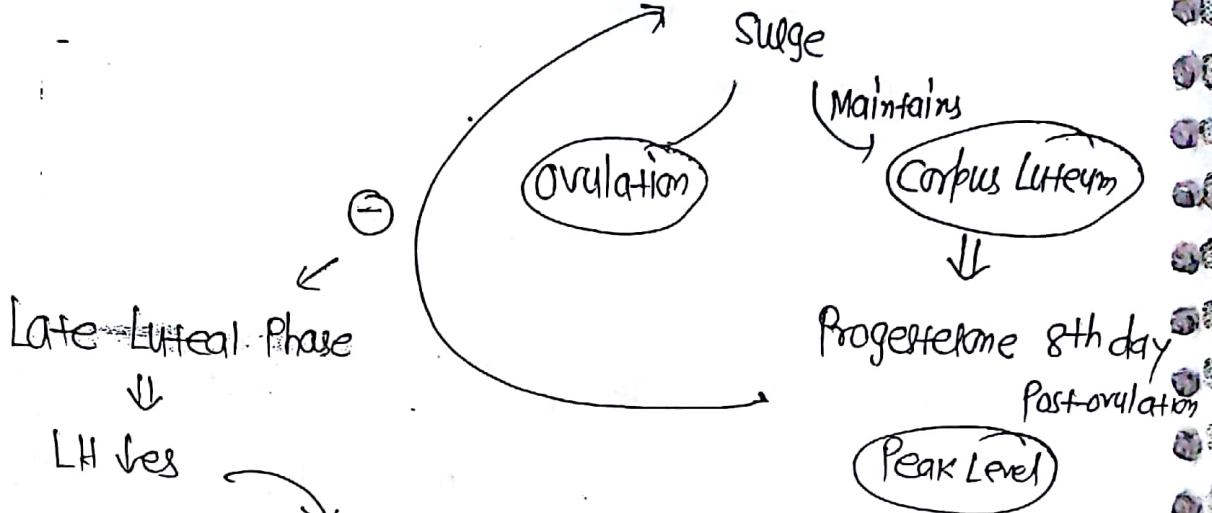
- (LH) ⇒ Pulse Frequency is high
- (FSH) ⇒ Low Pulse Frequency

# MENSTRUAL CYCLE



Ovary

Luteal Phase → Start ⇒ onset of LH



Menstrual blood is mainly Arterial

Spiral Arterioles  
 ↑ Vasoconstriction

Avascular ← Endometrium

Necrosis

↳ Shed's off (Menses)

Myometrium

↓

10 dysmenorrhoea (Physiological) → if present then ovulatory cycle ⊕

Anovulatory bleeding ⇒ Painless

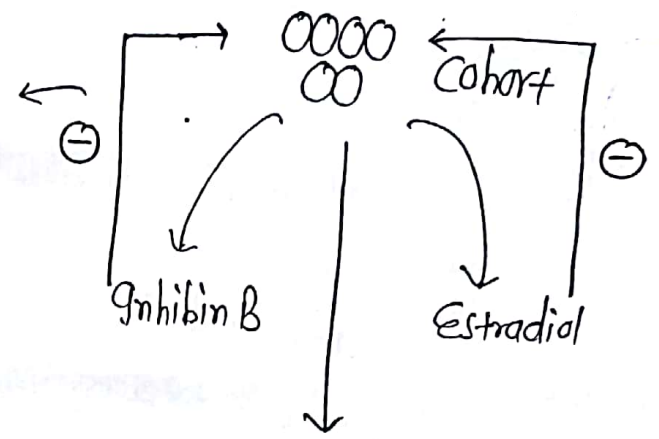
↓ Progesterone → GnRH (Small pulses) in Pulse frequency

(125)

Follicular phase

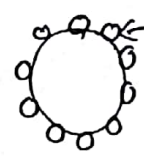
FSH

Controlled growth of follicle; so give ⊖ feedback to FSH



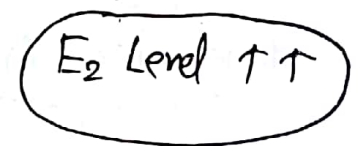
Dominant follicle selected

Middle Proliferative Phase



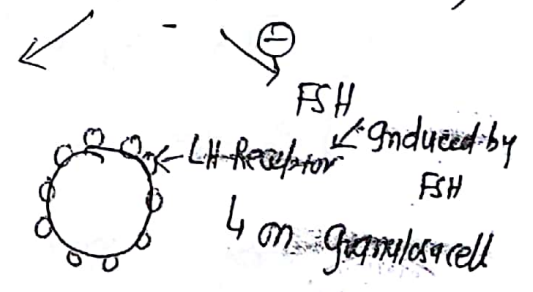
4m Dg

Late Proliferative Phase



High (200 pg x 48 hrs)

LH Surge



Luteinization of Granulosa cells



Testosterone → Sex Steroids

↓  
Intracytoplasmic (R)  
(In the absence of Ligand) C19

Major Source of testosterone in ♀ ⇒ i) Peripheral Conversion of (50%)  
Androstenedione

ii) 25% adrenal gland

iii) 25% ovary - theca cells  
↓ (theca interna)

- Ovary doesn't produce DHT (dihydro testosterone)
- Ovary also doesn't produce DHEAS sulphate

Androgen

↳ Which androgen is in Max<sup>m</sup> amount  
↓

Androstenedione > DHEA > Testosterone

Produced by Adrenal gland only.

\* Gene for Androgen (R) Located

↳ Long Arm of X-chromosome

\* Testosterone in ♀ ⇒ i) Puberche / Adrenarche <sup>def<sup>d</sup> Androgen</sup>

\* Estrog<sup>n</sup>

ii) Control of Libido

iii) Intraovarian Testosterone Level ↑

↓  
Antral Follicular Growth  
(Estrogen rich environment)

\* Testosterone in  $\sigma \Rightarrow$  Spermatogenesis (126)

$\Downarrow$   
Intra-testicular Level of testosterone is high

$\Downarrow$   
Sertoli cells produce TBP (Testosterone Binding Protein)

\* ~~Initiation of Spermatogenesis~~

$\hookrightarrow$  by ~~FSH~~

\* ~~Spermatogenesis Require~~  $\Rightarrow$  ~~FSH / Testosterone~~

\* Sertoli cells produce  $\Rightarrow$  ~~Müllerian Inhibiting substance~~  
~~TBP (Testosterone binding protein)~~  
Relaxin  
Inhibin  
Estradiol

\* Most of Testosterone is in Bound form: Binds to SHBG / Albumin  
 $\hookrightarrow$  1% Testosterone free (Male: 2% free)

\* Testosterone  $\xrightarrow{\text{Ove}}$  SHBG Synthesis  
 $\hookrightarrow$  takes place in Liver

Qo. Which has higher affinity to bind to SHBG?

~~Testosterone~~  $>$  Estrogen

\* End product of Testosterone  $\Rightarrow$  ~~Oxosteroids~~ (ketosteroids)

Q. Which cells form Blood-testis barrier

↳ Junction of Sertoli-Sertoli cells  
↳ Physical barrier b/w blood vessels & seminiferous tubules

2 Compartments : ADLUMINAL COMPARTMENT : 1<sup>o</sup> Spermatocyte  
2<sup>o</sup> Spermatozoa

Inner side of tubules;  
isolated from blood & lymph

Basal compartment : Spermatogonia

↳ Outer side of tubules; in contact  
w/ blood & lymph

\* Normal Menstrual cycle

↳ Length = 21-35 days

acc. to FIGO; Length = 24-38 days

Avg. Length  $\Rightarrow$  28 days

Amount of blood loss  $\Rightarrow$  80ml

Average of blood loss (amount)  $\Rightarrow$  35-50 cc

No. of days = 2-7 days

average No. of days =  $4\frac{1}{2}$  - 5 days

\* Abnormal Uterine bleeding (AUB)  $\Rightarrow$

Menorrhagia  $\Rightarrow$  More Amount ( $>80ml$ ) or More Menstrual day ( $\geq 8$  day)  
Length of cycle = (N)

Hypomenorrhoea  $\Rightarrow$   $<2$  day or  $<20ml$

Polymenorrhoea  $\Rightarrow$   $<21$  day ( $<21$ ) Length of Menstrual cycle

Oligomenorrhoea  $\Rightarrow$   $>35$  day ( $>38$ ) Length of Menstrual cycle



Metrorrhagia  $\Rightarrow$  Irregular bleeding / Intermenstrual

- classification of AUB acc. to FIGO  $\Rightarrow$  (127)

PALM - COEIN System  $\Rightarrow$

|                         |                                   |
|-------------------------|-----------------------------------|
| AUB P - d/t Polyp       | AUB C - d/t coagulation defect    |
| AUB A - d/t Adenomyosis | AUB O - d/t ovulatory dysfunction |
| AUB L - d/t Leiomyoma   | AUB E - d/t endometrial causes    |
| AUB M - d/t Malignancy  | AUB I - d/t Iatrogenic            |
|                         | AUB N - Not yet classified        |

$M_x \Rightarrow$  acc. to its cause

AUB  $\geq 45$  yr  $\rightarrow$  evaluated for Endometrial carcinoma  
(do Endometrial biopsy to Rule out Endometrial carcinoma)

## PUBERTY

- development of Secondary sexual character.

|                                                                     | <u>Girls</u>        | <u>Boys</u>         |
|---------------------------------------------------------------------|---------------------|---------------------|
| (N) Age $\Rightarrow$ of Puberty                                    | $10 \frac{1}{2}$ yr | $11 \frac{1}{2}$ yr |
| Precocious Puberty $\Rightarrow$<br>$\uparrow$ More common in girls | $< 8$ yr            | $< 9$ yr            |
| Delayed Puberty $\Rightarrow$<br>$\downarrow$ More common in boys   | $13$ yr             | $14$ yr             |



1st sign of Puberty

Girls  
Growth Spurt

Boys  
Testicular enlargement

1st visible sign of Puberty

Thelarche  
(Appearance of Breast bud)

Testicular enlargement

(Tanner stage - 2)

aa Growth Spurt → Thelarche



Pubarche  
(Adrenarche)



Peak Height Velocity → Breast



Tanner stage 3

Time gap = 6 months ← [ ↓  
Menarche

Testicular enlargement



Penile enlargement



Pubarche



Peak height velocity

\* M/c Cause of Precocious Puberty in girls ⇒ Idiopathic (90%)

so, girls come c Precocious Puberty  
evaluate c MRI brain

Brain tumor (10%)

↓  
M/c Brain tumor



Hemangioma

## Central Precocious Puberty

- Premature activation of HPO axis

LH/FSH  $\uparrow$

Isosexual

## Peripheral Precocious Puberty <sup>253</sup> (128)

Peripheral source of sex steroid hormones

Estrogen / ~~Progesterone~~ Androgen

↳ secreting tumours

M/c a/w  $\Rightarrow$  McCune Albright Syndrome  
(Precocious Puberty + Café-au-lait + Polyostotic fibrous dysplasia)

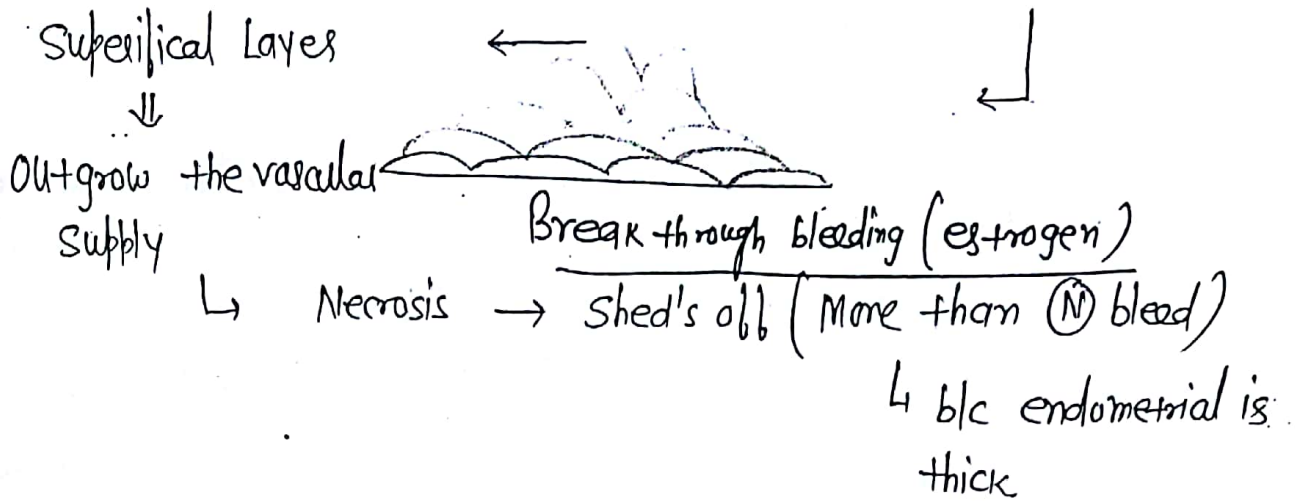
It may be isosexual or may be heterosexual.

- \* M/c cause of delayed Puberty in Males  $\Rightarrow$  Constitutional delay  
 $\Downarrow$   
Other Relatives have same history of delayed Puberty

\* Other problems in adolescent / Pubertal age group

↳ Irregular bleeding - M/c cause Anovulatory bleeding  
2nd M/c  $\Rightarrow$  Coagulation defects

\* In Anovulatory cycle → Unopposed estrogen



\* GOC ⇒  
for Adolescent  
Irregular bleeding

OCP

↳ It Regularise the cycle

It also less blood loss.

Protects from Unwanted Pregnancy  
also less Pelvic Inflammatory disease

also gives - only Progesterone pills

QA:

20 days bleeding → Unstable vitals

↳ So; endometrium is thinned at time of presentation

↓  
Initially give high dose i/v Estrogen

↳ do endometrium Proliferation  
quickly and stops bleeding by  
Sealing the breaks

↓  
Finally give Progesterone only pills / Combined ocp

\* High dose estrogen only given in unstable vitals;  
 b/c in all other cases estrogen also causes closure  
 of epiphyseal plate (stop Growth spurt). (129)

Least effective  $\Rightarrow$  Mefenemic Acid (NSAIDs)  
 $\Downarrow$   
 When hormonal therapy is Not taken  
 by girls.  
 Tranexamic acid (antifibrinolytic drug)  
 $\hookrightarrow$  Stop bleeding

### MENOPAUSE

- Cessation of Menses for 12 consecutive Months.
- Avg. Age  $\Rightarrow$  51 yr
- Avg. Age in India  $\Rightarrow$  47 yr
- It doesn't need any investigation; it is history based diagnosis.

\* Premature Menopause  $\Rightarrow$  < 40 yr  
 (Premature ovarian failure  
 1° ovarian insufficiency)

$\hookrightarrow$  Confirmed by SFSH Level: if it is  $\geq 40$  IU  
 on 2 occasions done 1 month apart - diagnosed  
 Premature Menopause

\* Delayed Menopause  $\Rightarrow$  doesn't happen by 55 yr of age  
 $\Downarrow$   
 Endometrial Evaluation (Endometrial biopsy)



FSH ↑

LH ↑

Estradiol → ↓ ( $< 20 \text{ pg}$ )

Testosterone production from the ovary continues just like before

Gradual Process (climatic phase)

↳ Hormonal changes start.  
Perimenopausal Phase / early Menopause

M/c Symptom ⇒ Vasomotor Symptom

↳ Hot flushes

↳ d/t Estrogen withdrawal

↳ coincides w/ LH Surge

Hot Flushes

⇒ Sudden feeling of warmth followed by } Last  
Diaphoresis } for  
1-5 min.

↳ More @ Night, (so; disturb sleep wake cycle)

↳ Moderate - Severe (disturbs daily routine)  
hot flushes

↳ to Start HRT (Hormone Replacement Therapy)

Q. 9/ patient has Intact uterus ~~But not hysterectomy~~

↳ give (E) + P → No Rde in Hot flushes

↳ Systemic therapy

(transdermal > oral)

Q. In Post-hysterectomy patient

only (E) (1st Line)

(130)

↓ if (E) is C/I

give SSRI (2nd Line)

3rd Line drugs →

clonidine  
Gabapentin  
Pregablin

In pre-menstrual  
Syndrome it is  
1st Line of drug

\* if patient is Not tolerating oral Progesterone

↳ give Mirena (LNG-IUD)

(E) + Bazedoxifene (SERM + SERD)

protects the  
Endometrium

Good effect on  
Bones

Other SERMs ⇒

M/C side effect  
↳ Hot flash

Tamoxifene  
Raloxifene  
Clomiphene  
Ormeloxifene

Not used in Rx of  
hot flash

Tamoxifene ⇒ given in Breast cancer patient  
↑ Risk of Endometrial cancer

Raloxifene ⇒ doesn't ↑ Risk of endometrial ca

↳ can be used for post-menopausal osteoporosis

\* Doc for Post-Menopausal osteoporosis  $\Rightarrow$  Bisphosphonates

ORMELOXIFENE  $\Rightarrow$  Centchroman (Saheli)

└ Indian government  $\Rightarrow$  Chhaya  
└ Non-~~steroidal~~ Steroidal contraceptive

It makes endometrium out of phase & prevents  
Implantation.

PERIMENOPAUSAL WOMEN  $\Rightarrow$  Cycle become Anovulatory; so;  
Irregular bleeding starts

DUB (Dysfunctional  
Uterine bleeding)

(Estrogen break through)

during the phase of  
active bleeding

43yr  $\pm$  3 Month Amenorrhoea

Bleeding  $\times$  20 days

Unstable  
vitals

$\Downarrow$

do D&C

Quickly stop

bleeding / in place  
of high dose estrogen

$\rightarrow$  An Adolescent: NO D&C b/c it alters the fertility

Stable  
vitals

$\Downarrow$

High dose oral

Estrogen  $\Rightarrow$  stop the

bleeding in 24 hrs

$\rightarrow$  alter that give  $\textcircled{P}$ /OCP

doesn't bleed from  $2\frac{1}{2}$  A

During phase of  
Amenorrhoea

$\Downarrow$

Gives OCP /  $\textcircled{P}$  alone

\* Any Reproductive age women; comes to H/O Amenorrhoea  
 Firstly Rule out Pregnancy.

(131)

V.V.G.\*\*\*

## AMENORRHOEA

### Primary

- Absence of Menses by 15yr  
 of age in the absence of  
 2° sexual character

OR

Absence of Menses by 15yr  
 of age in the presence of  
 2° sexual character

M/c Cause  $\Rightarrow$  Gonadal dysgenesis

2nd M/c Cause  $\Rightarrow$  Mullerian agenesis

### Secondary

- Absence of Menses for 90 days  
 in a previously menstruating female  
 (In Irregular cycles  $\Rightarrow$  6 Months  
 Absence of  
 Menses)

- M/c Cause  $\Rightarrow$  Pregnancy

- M/c Pathological cause  $\Rightarrow$  PCOS

## PRIMARY AMENORRHOEA

- Look about Thelarche

Absent

$\Downarrow$

d/t Gonadal dysgenesis  $\Rightarrow$  Gonads Abnormal

Kallman's Syndrome }  $\Rightarrow$  Gonads Normal  
 constitutional delay }

Present



# Gonadal dysgenesis (Raised LH; FSH)

- Turner
- Pure gonadal dysgenesis
- Mixed gonadal dysgenesis

## TURNER

Karyotype  $\Rightarrow$  XO

## PURE GONADAL DYSGENESIS

46 XY / 46 XX

$\hookrightarrow$  Swyer's Sx

(Mutation in SRY gene)

Non-functional

d/f absence of Y chromosome

Functional (SRY gene)

Gonads — ovary —

Internal Genitalia — Female —

absence of MIS / testosterone

Estrogen

$\downarrow$

Growth Puberty

$\rightarrow$  Absence

$\hookrightarrow$  Hypoplastic

External Genitalia — Female —

absence of DHT (active form of testosterone)

Gonads  $\Rightarrow$  Ovary

$\downarrow$

Accelerated Atresia

fibrosis (Fibrotic Streaks)

Fibrotic gonads

$\downarrow$

fibrotic ovary

Fibrotic / Nonfunctioning Streaks / testes

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TURNERPURE GONADAL  
DYSGENESISMIXED GONADAL  
DYSGENESIS

(32)

Uterus  $\Rightarrow$  present  
(Hypoplastic)

present

Mullerian / Wolffian  
both ducts  $\oplus$  int. b/c  
res. acts on i/L  
Mullerian duct

external  
genitalia  $\Rightarrow$  Female  
Like

Female  
Like

Ambiguous genitali  
(Like of both male & female)

Pubic /  
Axillary hair  $\Rightarrow$  present  
(Sparse  
Scanty)

present

Present

Breast  
development  $\Rightarrow$  Absent

Absent

Absent

Q.10. Most characteristic feature of Turner Syndrome  $\rightarrow$   
Short stature

Stature  $\Rightarrow$  Short stature

N/Tall

N/Tall

45 X 0

Absent (X)

$\downarrow$   
Absence of (SRY) gene

$\downarrow$   
Responsible for growth  
of long bone

# TURNER SYNDROME

↳ Short Stature

Short webbed Neck

Low Posterior hair line

M/c congenital heart disease  $\Rightarrow$  Bicuspid ~~heart~~ <sup>Aortic valve</sup>

Short 4th Metacarpel

Cubitus valgus (elbow deformity)

Rudimentary ovaries

No Menstruation

True about Turner's  $\Rightarrow$

~~(a) Uterus~~  $\oplus$  Breast  $\ominus$

(b) Both  $\oplus$

(c) Uterus  $\ominus$  Breast  $\oplus$

(d) Both  $\ominus$

\* I.Q.  $\Rightarrow$  (N) in Turner Syndrome

I.Q.  $\Rightarrow$  Ab. Sub Normal  $\Rightarrow$  when extra "X" chromosome

↳ Gn "Klinefelter Sx" (47XXY)

↳ M/c sex chromosome Abnormality.

\* Life-Span  $\Rightarrow$  Slightly less

\* LH/FSH  $\Rightarrow$  Yes (so; Turner Sx is klas "Hypergonadotropic hypogonadism")

## TURNER'S (45X0)

FSH ↑

Short Stature

Streak ovaries

Anosmia ⊖

Uterus ⊕

Breast development ⊖

External genitalia Female Like

Pubic/Axillary Sparse

## KALLMAN'S (46XX)

FSH ↓ (Hypogonadotropic hypogonadism)

Ⓝ / tall

Ⓝ ovaries

Anosmia ⊕

⊕

⊖

Female Like

Sparse

\* Constitutional delay

↳ Diagnosis of exclusion

Short @ Presentation

Ⓝ Karyotype

\* GOC for Primary Amenorrhoea ⇒ Karyotype

\* Rx for Gonadal dysgenesis ⇒ give E/P

↳ She doesn't Need Surrogate mother, Needs ovum donor



\* Primary Amenorrhoea  $\pm$  Secondary sexual characters

v/vg  
/ ii>

Mullerian Ageneis;

ii> AIS (Androgen Insensitivity Syndrome);

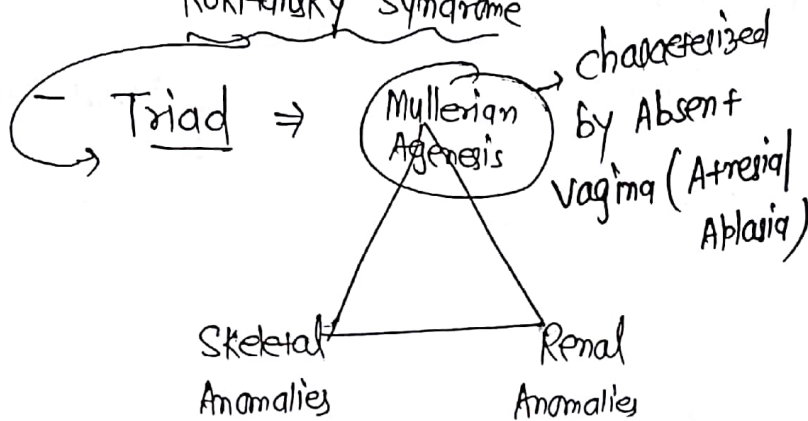
iii> Imperforate hymen;

iv> Transverse vaginal septum

Mullerian Ageneis

Klar "MRKH Syndrome"

Rokitansky Syndrome



Absent vagina

Absent uterus

Fallopian tube are absent - Proximally  
Present - Distally

- Gonads - ovary (N)

Karyotype - 46XX

AIS

- Klar "Testicular Feminization Syndrome"

- Testis is producing Androgen; but Receptor is completely insensitive

- Blind ending vagina (Small vaginal Pouch)

- Uterus Absent (b/c testis produce MIS)

- Testis (+) (functional)

Karyotype  $\Rightarrow$  46XY

## Mullerian Agensis

Breast development  $\rightarrow$  Present  
external Genitalia  $\rightarrow$  Female Like

AIS

265

(34)

Present @ Puberty

Female Like

Pt. May have Inguinal hernia & carrying Undescended testes

$\hookrightarrow$  May cause Malignancy

$\downarrow$   
do gonadectomy  $\Rightarrow$  after Puberty

$\downarrow$   
if done before Puberty;  
AIS patient Neither have female Nor Male; so done after Puberty

• Male Pseudohermaphrodite  
(M/c cause  $\Rightarrow$  AIS)

$\rightarrow$  Genotype Male; Phenotype Female

• Female Pseudohermaphrodite

M/c cause  $\Rightarrow$  CAH

$\hookrightarrow$  deficiency of enzyme 21-hydroxylase

Female phenotype  $\rightarrow$  Y chromosome

$\downarrow$

as soon as diagnosis is made  $\leftarrow$  Should undergo gonadectomy

Pubic /  
Axillary hair

Mullerian Ageneis  
(N)

AIS  
Absent (Sparse)  
Scanty

Pregnancy

Needs Surrogate Mother  
Generally - the child  
is of Mullerian  
Ageneis patient.  
(by GV technique)

Physically Absolutely  
(N); but they  
have worst Reproductive  
outcome  
↓  
do vaginoplasty

Best to  
differentiate

⇒

Karyotyping

S-testosterone  
Level

⇒

(N)

(N female Level)

Elevated  
(Like of Male Level)

Q:

After doing Karyotype in p Amenorrhoea; Next + test

USG (≡)

USG + FSH (≡)

\*

TRUE HERMAPHRODITE ⇒ Tissue of both ovary & testes  
(ovo-testes)

↳ also have Ambiguous Genitalia

Mosaic Karyotype

↳ S/O of Mixed gonadal dysgenesis ⇒ differentiate by  
HPE of ovary & testis

## IMPERFORATE HYMEN

- Outflow tract obstruction
- everything else — Karyotype — (N)
  - Internal genitalia — (N)
  - External genitalia — (N)
  - development of Puberty — (N)
- H/O  $\Rightarrow$  Primary Amenorrhoea ; cyclical abdominal Pain
- On Local exam<sup>n</sup>  $\Rightarrow$  bluish distended Membrane
- Sometimes pr<sup>t</sup>.  $\bar{c}$  Emergency  $\Rightarrow$  Acute Urinary obstruction
  - Hematometra }  $\Rightarrow$  Bladder outflow obstruction
  - Hematocolpos }

## TRANSVERSE VAGINAL SEPTUM

- M/c site  $\Rightarrow$  Upper 1/3rd of vagina
- H/H = cruciate incision — Impeforate hymen
- Excision — vaginal septum (transverse)



\* Reifeinstein Syndrome  $\rightarrow$  Partial Androgen Insensitivity Syndrome

Complete AIS  $\rightarrow$  Clitoris - hypoplastic  
Labia Majora - "

Partial AIS  $\rightarrow$  Clitomegaly seen

$\hookrightarrow$  Both girl looking or Boy looking Person May have Partial AIS (depend on how Much Receptors are Active)

\* Precocious Menarche  $\rightarrow$  Menarche starts <sup>without</sup> ~~before~~ 2° sexual character  
 $\hookrightarrow$  < 10yr Menarche starts

## SECONDARY AMENORRHOEA

• Rule out @

GOC! Hormone assessment

always together  $\leftarrow$   $\begin{cases} \text{TSH} \rightarrow \text{both hypo \& hypel thyroidism} \\ \text{Prolactin} - \text{Hyperprolactinemia} \end{cases}$

$\downarrow$   
b/c TRH is stimulatory to Prolactin

FSH ( $\pm$  LH) ( $\pm$  E<sub>2</sub>)

$\hookrightarrow$  M/C Cause

$\hookrightarrow$  Pituitary Microadenoma (< 1cm)

$\hookrightarrow$  M/C presentation

$\hookrightarrow$  Galactorrhea & Amenorrhoea

\* Prolactin One back GnRH

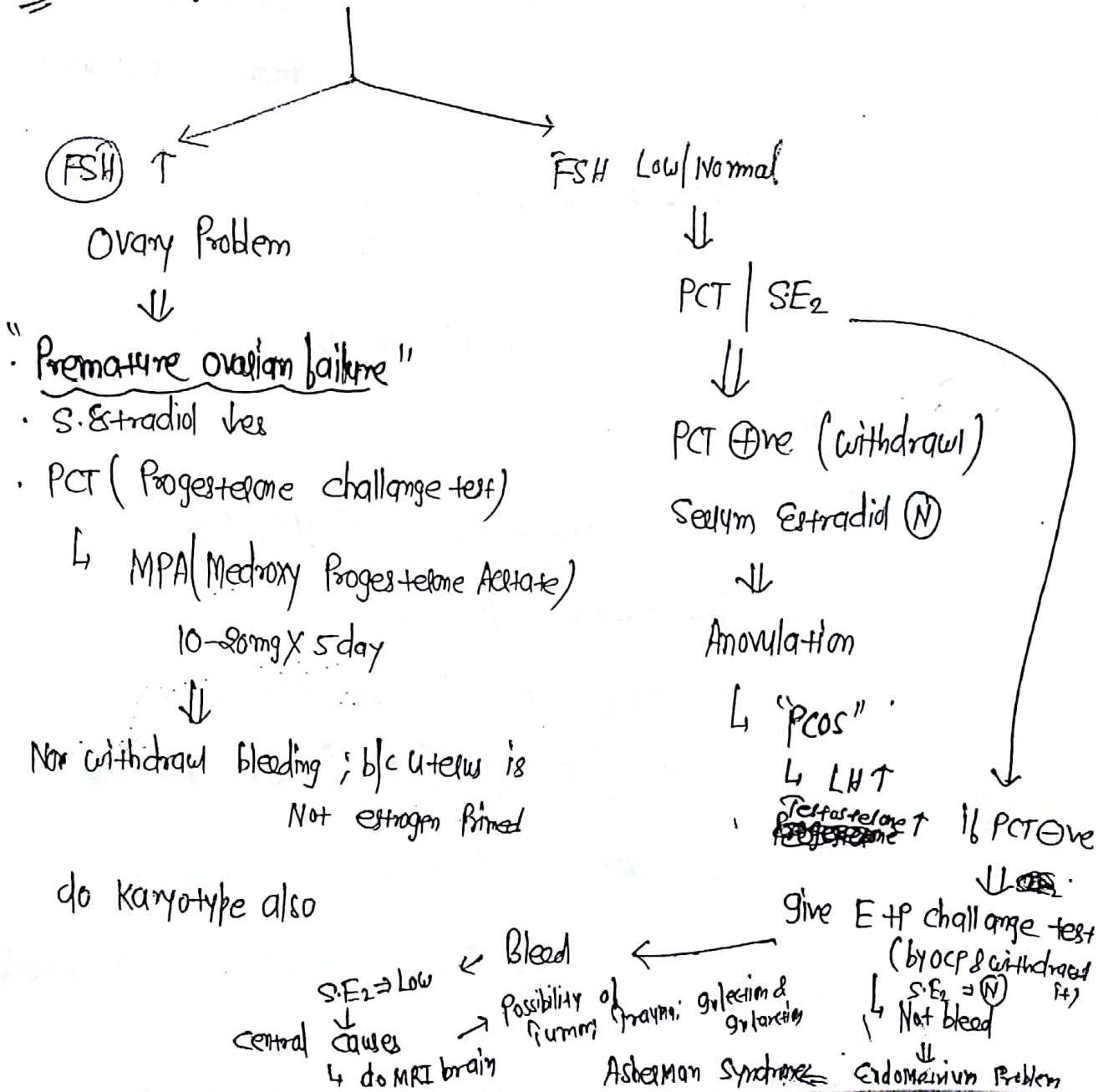
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↳ if  $< 50 \Rightarrow$  Repeat test  
 $> 50 \Rightarrow$  MRI

DOC for Prolactin  $\Rightarrow$  Cabergoline  $>$  Bromocriptine  
 (dopamine Agonists)  
 ↳ longer t<sub>1/2</sub>; so give twice a week only.

Q if TSH & Prolactin  $\Rightarrow$  (N)



MRI brain

↓  
(Normal)

↳ (Functional hypothalamic Amenorrhoea)

↓  
Diagnosis of exclusion

↳ Seen in Anorexia Nervosa

Stress Induced Amenorrhoea

Exercise Induced Amenorrhoea

Chronic Malnutrition

Tt of Premature ovarian failure

↳ HRT

(till the age of Menopause)

### ASHERMAN'S SYNDROME

• Intrauterine Adhesions

• M/c cause  $\Rightarrow$  vigorous curettage

• Highest Risk  $\Rightarrow$  PPH (vigorous curettage)

• M/c presentation  $\Rightarrow$  Menstrual Irregularities

↳ Amenorrhoea > Hypomenorrhoea

\* Single M/c Symptom/Presentation

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↳ Infertility.

\* Screening test  $\Rightarrow$  HSG (Not confirmatory test)

$\Downarrow$   
Adhesion seen as filling defect

In HSG

$\Downarrow$   
Multiple; Smooth;

Irregular Margin; Sma  
in look.

IOC - Hysteroscopy (confirmatory test)  
↳ both diagnostic & therapeutic

T/t  $\Rightarrow$

Hysteroscopic Adhesiolysis

+ Cu-T Insertion

$\Downarrow$   
to make distance b/w  
uterine wall

+ High dose Estrogen  
for 1 cycle

$\Downarrow$   
for quickly Proliferation

PCOS (Polycystic ovarian Syndrome)

- klas "Stein-Levinthal Syndrome" \*

- Primary Pathology lies in the ovary.

Stromal hyperplasia  
(Theca cells)



↑ Testosterone production  
↓ Anovulation



- ↑ Testosterone Androgen production ; Not only inside ovary ; but also in Periphery

↳ Symptoms (Hirsutism)

↳ also cause dyslipidemia

- 50% of girls have obesity (also produces Excess Unopposed Estrogen)  
↳ bc in Periphery  
Androgen  $\xrightarrow[\text{cell}]{\text{Aromatase}}$  Estrogen

- 75% of girls have Insulin Resistance

↳ causes hyperinsulinemia

Syndrome X  
(Metabolic syndrome)

↳ Hyperinsulinemia  
↑ Androgen production  
dyslipidemia

• ↑ Testosterone Androgen production  
• further  
• dyslipidemia

\* These patient have Resistance Test in LH

↳ Testosterone Androgen production

\* HAIR AN → Acromegaly ? ⇒ Cutaneous Marker of Insulin Resistance  
Hypertrophy     Insulin Resistance  
Androgenism     L Seen @ Nape of Neck  
Axilla  
Groin

## Rotteldam's criteria for diagnosis $\Rightarrow$

if 2 or more of the following criteria are Met Provisional diagnosis of PCOS;

i) Amenorrhoea and/or oligomenorrhoea  
 $\hookrightarrow$  d/t Anovulation

\* Menorrhagia  $\Rightarrow$  can be presentation (Mainly in obese pati)  
 $\hookrightarrow$  Not a criteria of Rotteldam's.

ii) Hyperandrogenemia and/or Hyperandrogenism

$\Downarrow$   
 High blood Level

$\hookrightarrow$   
 Total S. testosterone  $\uparrow$  (mildly elevated)

(N)  $< 70 \text{ ng/dl}$

PCOS =  $70 - 150 \text{ ng/dl}$

definitely  $< 200 \text{ ng/dl}$

if  $> 200 \text{ ng/dl}$  (severe) = Testosterone  
 secreting tumor

$\Downarrow$   
 Clinical feature

$\downarrow$   
 Hirsutism  $\rightarrow$  on chin, chest, Axilla, Thigh hair growth

Test terminal hair growth  
 in Male pattern distribution

ii) Acne - Resistant to  
 usual t/t and  
 Scarring in Nature

iii) Alopecia

\* Scoring System to confirm the Hirsutism

$\hookrightarrow$  Ferriman Gallway score  $\geq 8$

Not in PCOS  $\leftarrow$  Virilization (Hyperandrogenism)  
 $\hookrightarrow$  Severe testosterone - seen in Androgen tumor

Virilization presents  $\bar{c}$  :

- i) clitoromegaly
- ii)  $\uparrow$  Muscle Mass
- iii) Hoarseness of voice
- iv) Male pattern balding  
(Temporal Recession)

ii) USG criteria of Rotterdam's

a)  $\geq 12$  follicles in the ovary & each follicle  
 $< 10$  mm in size

And/or

b) Ovary volume  $\geq 10$  cc

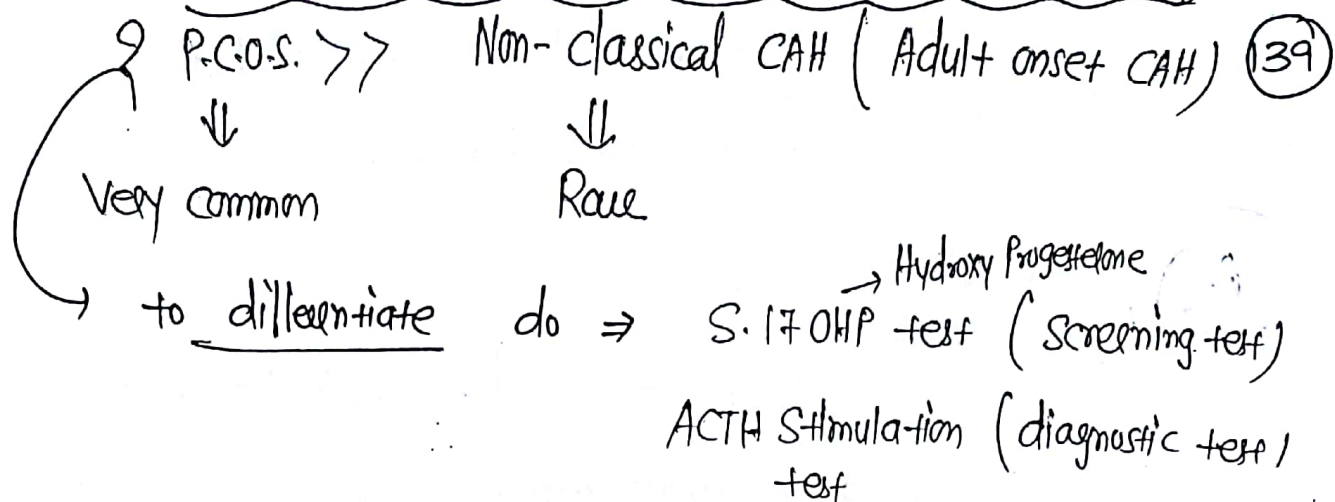
\* (N) Female ~~also~~ May have USG pictures of PCOS;  
(20-25+)

PCOS Female may have Normal USG pictures

\* if Follicles seem large in USG  $\Rightarrow$  Hyperstimulatory ovary.  
( $> 1$  cm)

\* In USG of PCOS  $\Rightarrow$  Necklace Pattern  
of ovary  
 $\downarrow$   
Characteristics to PCOS  
 $\swarrow$   
Not Rotterdam's criteria  
 $\hookrightarrow$  also Stromal hyperplasia  
Thick theca

\* If 2 criteria are Met; then Provisional diagnosis of  $\Rightarrow$



Other d/d of PCOS  $\Rightarrow$  Androgen tumor

$\hookrightarrow$  Gn it Rapid onset  
 Rapid progression  
 Testosterone  $> 200$   
 Virilization

Cushing syndrome

\* Lab Investigation  $\Rightarrow$  i) S. testosterone  
 (DHEAS - May be slightly elevated  
 $< 700 \text{ Ngm}$ )

ii) USG

iii) LH  $\uparrow$  / FSH (N)

$\hookrightarrow \frac{\text{LH}}{\text{FSH}} \text{ Ratio} \Rightarrow \text{Tes} (> 2:1)$

iv) T. Estrogen  $\uparrow$

Total  $E_1 \uparrow$  /  $TE_2 = (N)$   $\left\{ \begin{array}{l} \frac{E_2}{E_1} \text{ Ratio Reverse} \\ \text{Free } E_2 \uparrow \end{array} \right.$



v) SHBG ves (So; Free  $E_2$  Test)

↳ b/c testosterone Inhibits SHBG Synthesis  
↳ HypoGonadism.

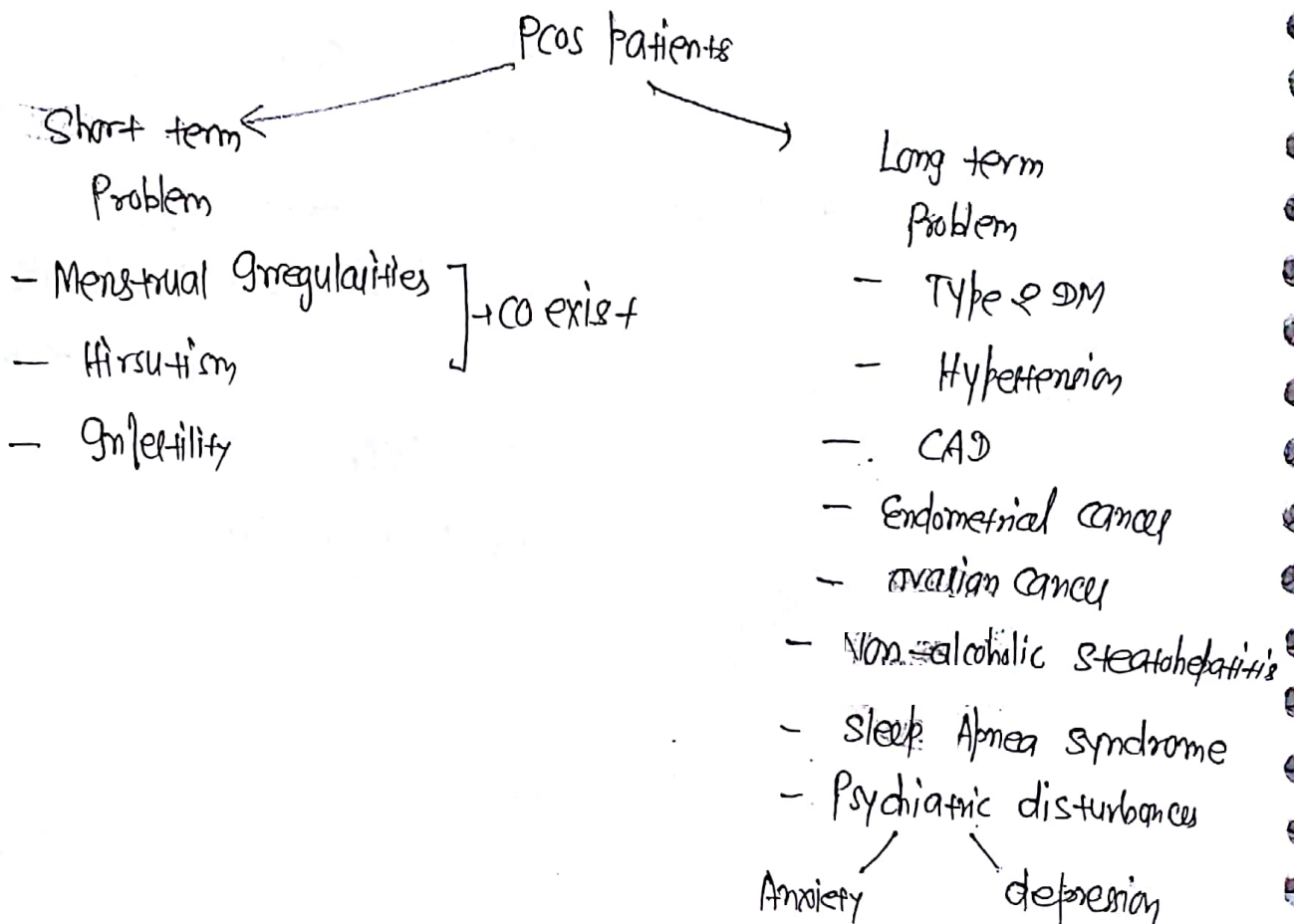
vi) Lipid Profile

vii) OGTT (to look for Insulin Resistance)

• DO  $\frac{\text{Fasting Glucose}}{\text{Fasting Insulin}} < 4.5$

viii) TSH } (N)  
ix) Prolactin }

x) 17 Hydroxy Progesterone test.



# - Pregnancy Complication

↳ Abortion

Gestational DM

Pre-eclampsia

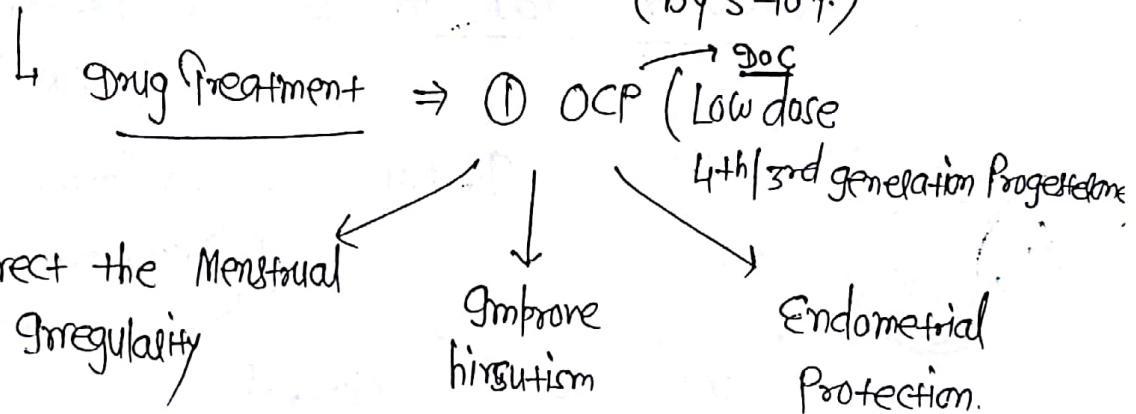
Pre-term-Labour

Still-birth,

(140)

## \* Management ⇒ Menstrual Irregularities + Hirsutism

Obese PCOS Patient → Advice weight loss  
(by 5-10%)



Doc for PCOS ⇒ OCP

## \* Only Hirsutism patient ⇒ give Spironolactone derivative

↳ Advice Not to conceive  
b/c it is Teratogenic drug

\* Cyproterone Acetate

\* Flutamide

\* Finasteride

\* Ketoconazole

\* GnRH Agonist

\* Metformin

Insulin sensitizer → weight loss (obese)  
 Can cause Lactic Acidosis  
 do LFT/KFT

## Eflornithine (Topical)

all drugs are given in Hirsutism except  $\rightarrow$  Danazol  
(Androgenic/E)

T/T for Infertility  $\rightarrow$  d/t Anovulatory

Obese PCOS  $\rightarrow$  weight loss

(weight gain is also advice in less weight patient)

Ovulation Induction  $\Rightarrow$  Clomiphene citrate  
(SERM) does antagonistic activity

It has . Zn component

(En) component

MOA  $\Rightarrow$  Central

Estrogen Receptor on pituitary prevents feedback inhibition



FSH  $\uparrow$  (FSH/LH)

Starting dose 50mg D<sub>2</sub>-D<sub>6</sub> / D<sub>5</sub>-D<sub>9</sub> of Menstrual cycle

Max<sup>m</sup> (Approved) drug  $\Rightarrow$  100mg.

L by FDA

Max<sup>m</sup> approved times  $\Rightarrow$  12 Months.

7% women ovulate after clomiphene  $\Rightarrow$  80% (141)  
 + women conceive  $\Rightarrow$  40%

Antagonistic effect  $\left\{ \begin{array}{l} \text{Cervix Mucus} - \text{Thick (Impermeable)} \\ \text{Endometrium} - \text{growth is affected} \end{array} \right.$

\* Poor Response (Not ovulating)

Obese patient (Insulin Resistance) - Clomiphene citrate + Metformin  $\rightarrow$  Clomiphene citrate (CC) also

ACOG obese PCOS patient  $\Rightarrow$  Doc for Infertility

2.5mg  $\longrightarrow$  7.5mg (Aromatase Inhibitor)  $\left\{ \begin{array}{l} \text{Letrozole (Not given for everyone; b/c Not FDA approved)} \end{array} \right.$

\* Clomiphene citrate  $\rightarrow$  Not teratogenic

M/c Side effect of clomiphene citrate  $\Rightarrow$  Hot flushes

2nd M/c  $\Rightarrow$  ovarian cyst formation

Other side effect  $\Rightarrow$  Multiletal pregnancy

(6-8%)

$\rightarrow$  only twins

(Not higher order gestation)

$\Rightarrow$  OHSS (ovarian hyperstimulation syndrome)

$\rightarrow$  1% (Negligible)



## 2nd Line drug for ovulation induction $\Rightarrow$

- Injection Gonadotropin (preferred)  
(FSH/LH)

- Laproscopic Ovarian  $\Rightarrow$  S/E  
drilling  $\hookrightarrow$  Premature ovarian failure

- Inj. HMG

(Human Menopausal Gonadotropin)

$\hookrightarrow$  taken from Urine of Post-Menopausal women.

- Recombinant Purified  $\rightarrow$  Potent  
preparation Inj. FSH

Highly expensive

More S/E as well

$\hookrightarrow$  Multiletal - 30% higher  
Pregnancy order Gestation

$\hookrightarrow$  OHSS - 15%

### \* Step up Protocol

Start  $\bar{c}$  Low dose



Monitor Response

$\hookrightarrow$  rising the dose

## 3rd Line drug for ovulation induction

Doc of ovulation induction  
in hypothalamic cause/Kallmann's Syndrome  $\Rightarrow$  GnJ. GnRH  
 $\downarrow$   
Pulsatile

\* Multifetal  $\Rightarrow$  Gonadotropin > clomiphene > GnRH Agonist  
♀ Risk Citrate

\* OHSS Risk  $\Rightarrow$  Gonadotropin > GnRH > clomiphene > GnRH  
Agonist Citrate Antagonist  
 $\downarrow$   
Prevention of  
OHSS.

In general  $\Rightarrow$

Bromocriptine  $\rightarrow$  Anovulation

↳ d/t hyperprolactinemia

Aromatase Inhibitors  $\rightarrow$  Letrozole

# \* OVARIAN HYPERSTIMULATION SYNDROME (OHSS)

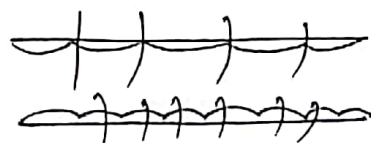
R/F  $\Rightarrow$

- i> Younger Age;
- ii> PCOS;
- iii> High Serum  $E_2$  Levels ( $>3500$  pg)
- iv> Large size of follicle & Large No. of follicle ( $>20$  follicles &  $>10$  mm in size)
- v> Pregnancy
- vi> Gonadotropin

Causes  $\Rightarrow$

Injection hCG (Used as ovulation Trigger);

• Mediator  $\Rightarrow$  VEGF  $\Rightarrow$  Cause Endothelial

 Injury  $\rightarrow$  Hypercoagulable state  
 $\hookrightarrow$  Leakage of fluid in 3rd space

Early - Clin 9 days of Inj. hCG

Late -  $>9$  days of Inj. hCG

$\hookrightarrow$  d/f Pregnancy Late OHSS seen

Prevention  $\Rightarrow$  Delay the hCG Injection;

called as "coasting"

$\leftarrow$  Cancel the cycle & do cryopreservation of embryo;

GnRH Antagonists

Volume expanders

Bromocriptine

# INFERTILITY

- Inability to conceive with one year of Unprotected intercourse
- But; if the patient is  $>35$ yr  $\Rightarrow$  Inability to conceive  $\geq 1$ m  
6 Months of Unprotected intercourse

Female factor contributes 40-55%

Male factor  $\xrightarrow{\text{"}}$  40%

Unexplained  $\xrightarrow{\text{"}}$  10%

Female Factor  $\Rightarrow$

Ovulatory Factor - 30-40%

Tubal Factor - 20-30%

Uterine Factor - 15%

Cervical Factor - 5%

Unexplained - 10%

$\rightarrow$  Ovulatory Factor  $\Rightarrow$  Most Reversible

$\hookrightarrow$  dlt Anovulation  $\xrightarrow{\text{do}}$  ovulation Induction

Premature ovarian failure  $\xrightarrow{\text{do}}$  donor ovum

Test of ovulation  $\Rightarrow$

i) Cervical Mucus

ii) vaginal cytology  $\rightarrow$  Lateral vaginal ferning  
(upper 1/3rd of lateral wall)

iii) Basal body temp. (Progesterone  
(BBT))

$\hookrightarrow$  Test BBT by  $0.5-0.8^{\circ}\text{F}$

if we plot Bimodal graph - cycle is ovulatory



iv) easiest test  $\Rightarrow$  S. Progesterone

$\Downarrow$

on D<sub>21</sub>  $\rightarrow \geq 3\text{ng} \Rightarrow$  ovulatory cycle

v) Best test — Endometrial Biopsy

$\Downarrow$

do in Premenstrual phase-

2 days before the expected date of Menstruation.

if endometrium Report  $\Rightarrow$  Secretory Endometrium  
 $\hookrightarrow$  ovulatory cycle

Proliferative Endometrium

$\hookrightarrow$  Anovulatory cycle, Telescopic gland  
 $\hookrightarrow$  See Long tubular glands & Pseudostratification  
 $\hookrightarrow$  dlt estrogen hormone

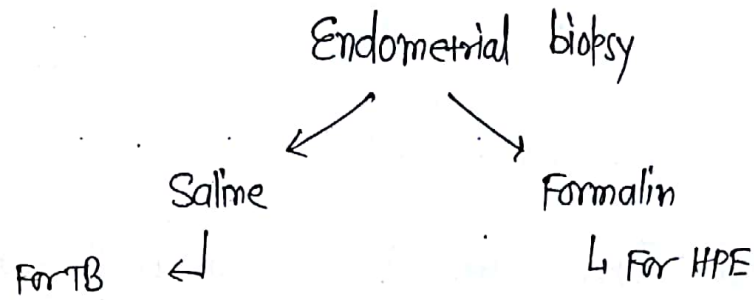
While Subnuclear vacuolation  $\rightarrow$  is the evidence of Progesterone Secretion on D<sub>16</sub> on HPE

Cork-screw glands  $\rightarrow$  Seen on D<sub>20</sub> on HPE (Late Secretory phase)

Max<sup>m</sup> Stromal edema  $\rightarrow$  Seen on D<sub>22</sub> on HPE

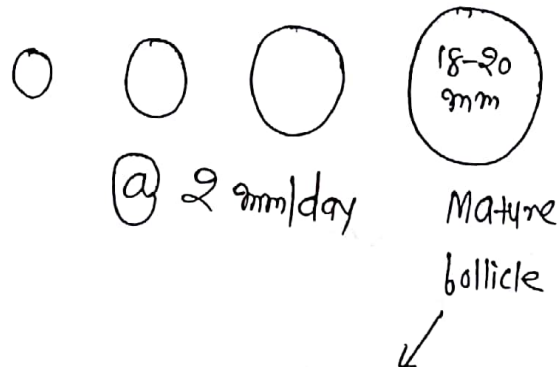
Leucocytic Infiltration of Endometrium  $\rightarrow$  (Premenstrual phase) on D<sub>26</sub> on HPE

\* Endometrial biopsy at least once in evaluation of women Infertility to Rule out Genital TB; (144)



vi) M/C used test of ovulation in Infertile Patients TVs for follicular Monitoring  $\Rightarrow$

Starts on D<sub>10</sub>



tells the Infertile patient to Intercourse.

$\rightarrow$  ① Sudden lvs in size and free fluid in Pouch of Douglas  
 $\Downarrow$   
Suggestive of ovulation.

- also in endometrium on USG  $\Rightarrow$

Trilaminar Endometrium  
(Triple Layer Endometrium)

$\hookrightarrow$  Three hyperechoic lines seen in Endometrium

$\hookrightarrow$  seen in "Periovulatory Phase".

\* Single hyperechoic line  $\bar{c}$  Posterior Enhancement

$\hookrightarrow$  d/t Secretion from Endometrium  
 $\hookrightarrow$  Suggestive of Secretory phase

vii) Urinary LH kits  $\rightarrow$

Urinary<sup>\*</sup> LH Surge  $\rightarrow$  After 24 hrs  
L ovulation takes place

### OVARIAN RESERVE

M/c used test for it  $\Rightarrow$  i) S. FSH  $\Rightarrow$   $\mathcal{D}_2 - \mathcal{D}_4$   
L on  $\mathcal{D}_3$  (Best)  
N value of S. FSH on  $\mathcal{D}_3$   
L  $< 10 \text{ IU}$

10-15 IU  $\Rightarrow$  Borderline Reserve

$> 15 \text{ IU} =$  Poor Reserve

7-20 IU = Suggestive of Premature ovarian failure

$\geq 40 \text{ IU} =$  diagnosis of Premature ovarian failure

ii) S. Inhibin B  $\Rightarrow$  on  $\mathcal{D}_3$  -  $< 45 \text{ pg}$  - Poor Reserve

iii) AFC (Antral Follicle count) - on  $\mathcal{D}_2 - \mathcal{D}_4$   
 $< 10$  follicle  $\Rightarrow$  Poor Reserve

iv) Best test  $\Rightarrow$  S. AMH (Anti Mullerian hormone; equivalent to MIS)  
Very small amount - Small prenatally follicle  
 $< 0.5 \text{ ng}$  - Poor Reserve  
 $\rightarrow$  b/c No fluctuation in menstrual cycle  
(False test - less)

✓ CCT (clomiphene citrate challenge test)

D<sub>3</sub> - S. FSH.

D<sub>5</sub>-D<sub>9</sub> - Clomiphene citrate 100mg

D<sub>10</sub> → S. FSH

High basal level which rise further on D<sub>10</sub> - poor Reserve

### TUBAL FACTOR



Fallopian tube should be patent.

GoC for Patency ⇒ HSG (Screening test)

↓ if abnormal

Best → Laparoscopy + Chromopertubation (Diagnostic)

Post-Menstrual Phase ⇒ D<sub>5</sub>-D<sub>11</sub> day (M/c on D<sub>10</sub>)

↳ d/t Cervix dilation

⊕nt @ this time

and also Pregnancy Ruled out @ this time

Peritoneal spill



cannula - Leech wilkinson's cannula



↓  
10mL dye (Water-soluble iodinated dye) used



- \* C/I of HSG  $\Rightarrow$
- ① Suspected  $\varnothing$  (UPT  $\rightarrow$   $\ominus$ ve);
  - ② known case of Genital TB;  
(Endometrial biopsy for Acid fast bacilli  $\Rightarrow$   $\ominus$ ve)
  - ③ Actively bleeding;
  - ④ current Pelvic Infection;
  - ⑤ K/c/o dye allergy

HSG as screening for  
Uterine Pathologies

- Adhesion
- Polyp
- Submucosal fibroid
- Mullerian Anomalies

Other Screening  
Modality

$\Rightarrow$

Sono hystelography

$\hookrightarrow$  Uses USG & Normal saline

B/L cornual block (Proximal block)

B/L Hydrosalpinx  $\Rightarrow$  happen d/t distal block

$\hookrightarrow$  Represents severe injury

B/L Proximal block  
Mid segment block  
distal block

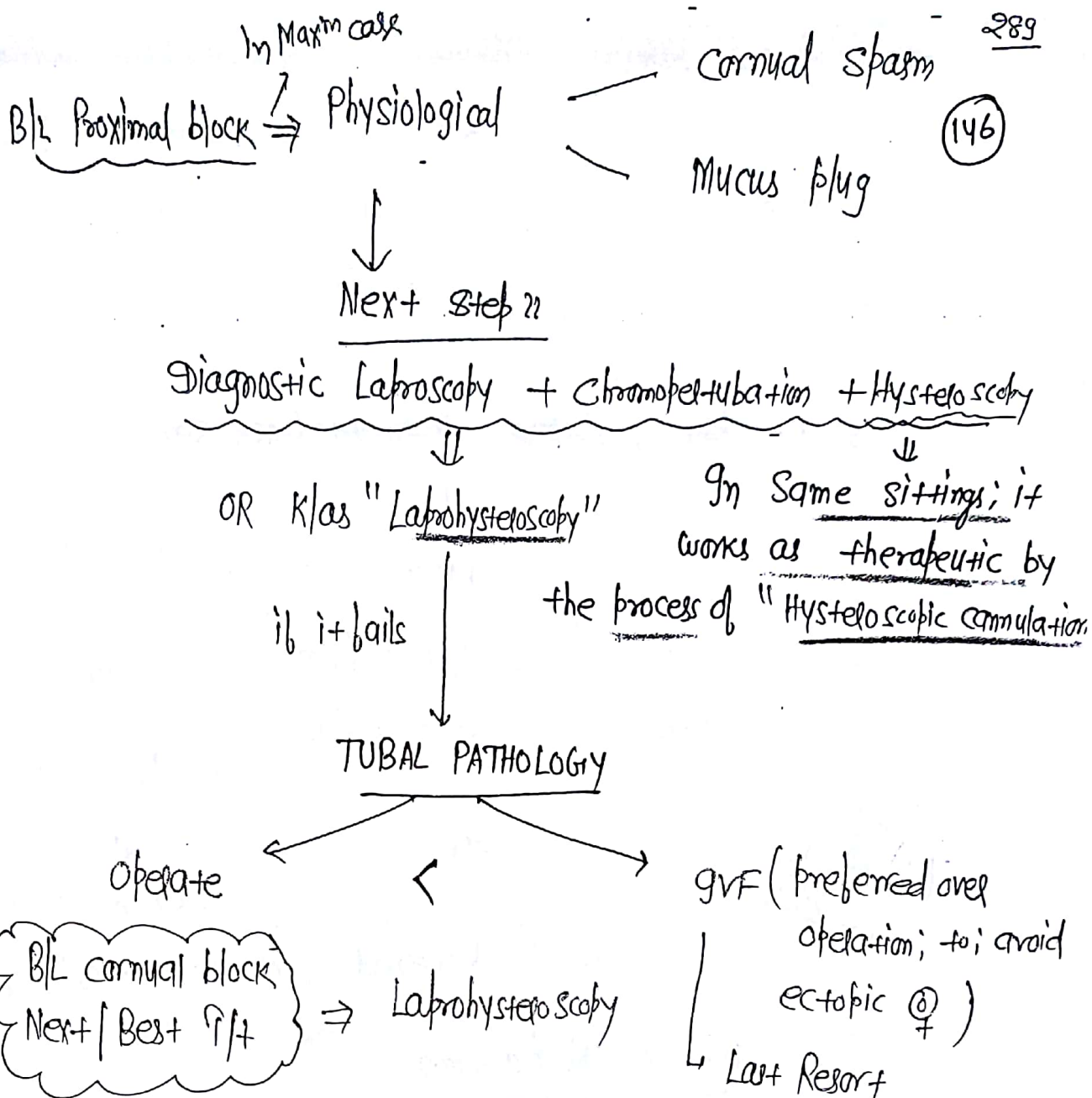
}

$\Rightarrow$

Best prognosis

$\hookrightarrow$  B/L Proximal block

$\hookrightarrow$  very less pathologically



\* if on HSG; B/L distal block seen

$\Downarrow$   
do Diagnostic Laproscopy

Mild  
 $\Downarrow$  if/it by  
Fibriolysis  
Adhesiolysis

Severe (B/L hydrosalpinx)  
 $\Downarrow$   
do gVF  
Fluid  $\Rightarrow$  embryo-toxic  
So; before gVF; do  $\Rightarrow$   
B/L Salpingectomy &  
Corneal clipping.

\* if on HSG; Proximal & distal block both seen on both of tubes separately (severe)



do gvf.

\* if on HSG; Mid segment-block seen



Tubal Ligation → Tubal Recanalization.

Good Prognostic Marker of conception after Tubal Recanalization → i) Type of Ligation

Clips > Falope Rings



Can be Reversed

can't be Reversed; Cauteary > Modified Pomeroy

ii) Type of Recanastomosis

Best ⇒ Isthmo-isthmic > Isthmo-ampullary

iii) Total Length after Recanastomosis > 4cm



iv) No other cause of Infertility

Test done before Anastomosis ⇒ Semen Analysis of husband

## GENITAL TUBERCULOSIS

291

(147)

- Secondary Infection;
- M/c Primary - Lungs > Lymph Node
- M/c Route - Hematogenous  
Direct  
Lymphatics -  
Ascending Infections
- M/c site  $\Rightarrow$  Fallopian tube > 90%  
Endometrium 50-60%
- M/c Route  $\Rightarrow$    
Direct Spread
- L/c site  $\Rightarrow$  Vagina & vulva - <2%
- Fallopian tube - Acute - Red Inflamed edematous  
Chronic - Thick walled / Adhesions
- Cobble stone appearance on HSG  
Tobacco Pouch appearance on HSG  
Lead pipe or pipe stem appearance on HSG  
Beads on string appearance on HSG  
Golf club appearance on HSG  
B/L Hydrosalpinx on HSG  
  $\Rightarrow$  Genital TB



\* M/c site affected  $\Rightarrow$  Ampulla (1st site to be affected on Genital TB)

L/c site affected  $\Rightarrow$  Infected

Endometrium  $\rightarrow$  acute - Normal

$\hookrightarrow$  affects only superficial part

Myometrium - spared

Chronic - Adhesion / Ulcers  
(Asherman's Syndrome)

\* M/c Presentation  $\Rightarrow$  • Infertility (world over = 10%  
India = 17%)

• Pain

• Menstrual Irregularity

First Menstrual  
Irregularity

$\downarrow$   
Menorrhagia

More common

Menstrual Irregularity

$\downarrow$   
Amenorrhoea > oligo

\* M/c Finding in Reproductive Age

$\hookrightarrow$  (N) Pelvic exam

$\hookrightarrow$  Tenderness sometime

L/c Finding in Reproductive Age

$\hookrightarrow$  B/L Adnexal Mass

B/L Adnexal Mass  $\Rightarrow$  M/c finding in adolescent girls  $\pm$  Genital TB 293  
(148)

Diagnostic  $\Rightarrow$

- $\rightarrow$  Endometrial Biopsy (Best)
- $\rightarrow$  Menstrual blood PCR Analysis  
(1<sup>st</sup> day sample taken to check superficial layer of endometrium)

R<sub>x</sub>  $\Rightarrow$  ATT (Anti-tubercular therapy) X 6 Months

$\hookrightarrow$  Improve fertility status - Yes.

\* In case there is severe Tubal Pathology  
(distort Normal Anatomy)

$\hookrightarrow$  ATT doesn't improve fertility status;  
Go for IVF; but ATT is given  
for Genital TB

### UTERINE FACTORS

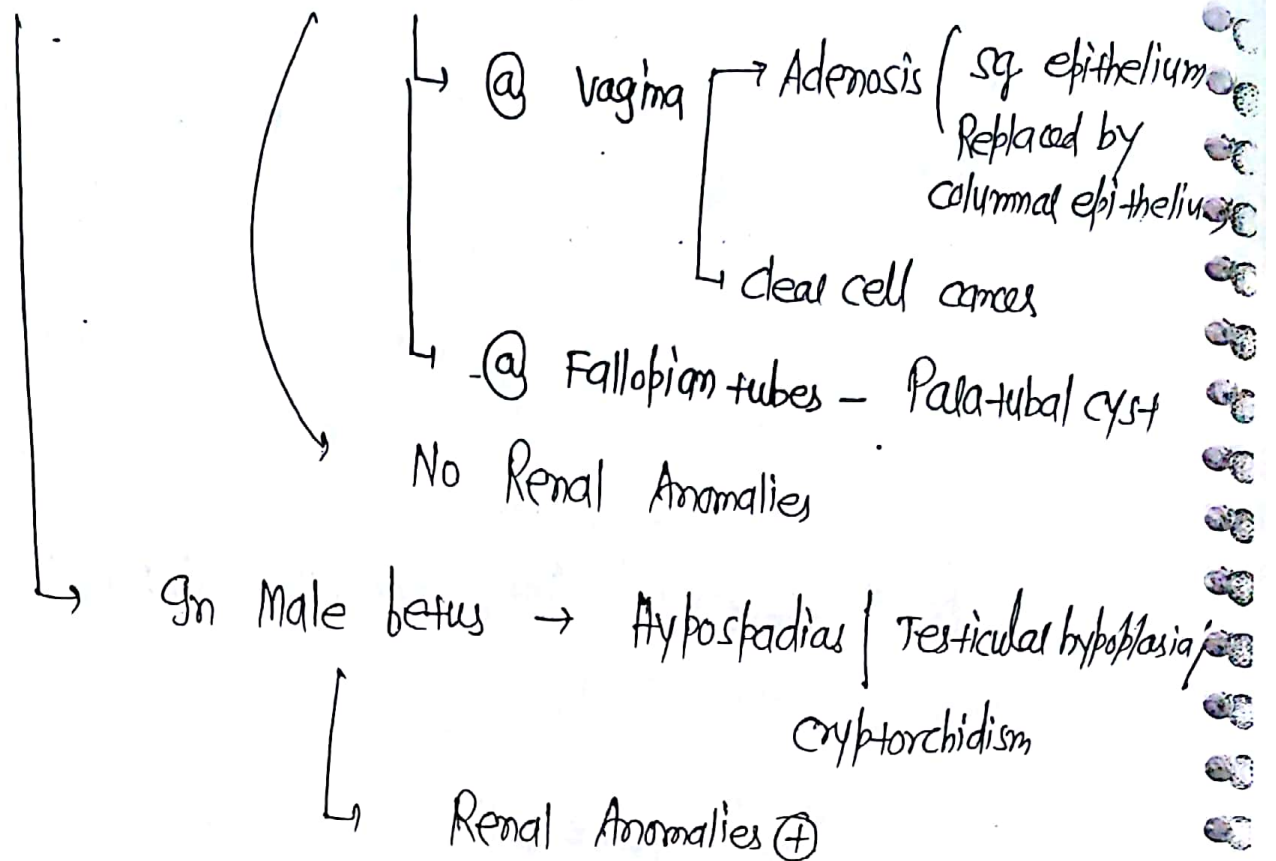
Infertility caused by  $\Rightarrow$

- i> Fibroid - Submucosal;
- ii> Polyp
- iii> Endometritis;
- iv> DES (Preg. women)

$\hookrightarrow$  Fetus Female  
 fetus

$\rightarrow$  Uterus (Hypoplasia)  
Most characteristic  
T-shaped uterine  
cavity

DES — In female fetus



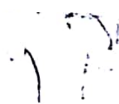
v) Acutely Retroverted Uterus

cochleate uterus ⇒ Acutely Anterverted Uterus

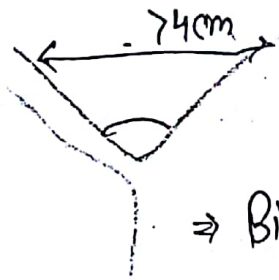
vii) Mullerian Anomalies

↳ M/c cause of Infertility — Septate uterus  
M/c cause of Abortion  
M/c Mullerian Anomaly  
Worst Reproductive Outcome

Best outcome ⇒ Arcuate > didelphys > Bicornuate



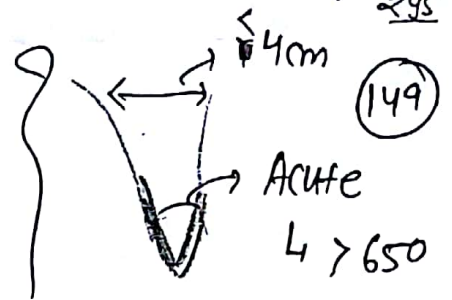
Screening test  $\Rightarrow$



$\Rightarrow$  Bicornuate

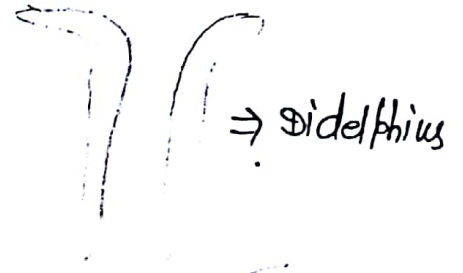
HSG  
(Non-diagnostic)

Septate  
uterus  $\Leftarrow$



old of septate uterus on HSG

$\hookrightarrow$  Bicornuate uterus



$\Rightarrow$  Didelphys

IOC  $\Rightarrow$  MRI <sup>\*\*\*</sup>  $\Rightarrow$  if showing fundal dip  $> 1\text{cm}$   
 $\Downarrow$   
Bicornuate.

Sonohysterography

3D/4D HSG (Not 2D USG)  
 $\hookrightarrow$  usually do

Gold Standard Investigation  $\Rightarrow$  { Laproscopy + hysteroscopy }  
 $\downarrow$   
alone is Not a good Modality.

\* All patients of Mullerian Anomalies Undergo evaluation  
for  $\rightarrow$  Urinary tract anomalies (Renal USG / xray)  
 $\Downarrow$   
High Risk



\* ectopic ovary  $\Rightarrow$  <sup>q/w</sup> Unicornuate Uterus  $\rightarrow$  U/L dysmenorrhea  
 $\rightarrow$  highest association  $\bar{c}$   
Urinary tract anomalies

\* TOC for Septate Uterus

$\hookrightarrow$  Hysteroscopic Resection  
(Tompkins / Jones)

### CERVICAL FACTORS

- Mucous affects fertility

$\hookrightarrow$  Characteristics — Impermeable to sperms  
 $\hookrightarrow$  Anti Sperm Antibodies — Post coital test <sup>r' klas " Sims Huhner test</sup>

$\hookrightarrow$  Post coital Cx Mucus — observed Under Microscope

Normal  $\rightarrow$  Forward Motility

ASA  $\oplus$  — AbN Motility

circulatory }  
shaky }

done @ 12-14th day of cycle

\* Newer test  $\Rightarrow$

Immuno bead Assay  
Sperm Agglutination  
Sperm Immobilization

Not doing in  
Current clinical  
Practice.

\* Tx of ASA

$\Rightarrow$  i) Intrauterine Insemination (IUI)

$\hookrightarrow$  Put sperm directly into  
uterus; bypass the Cx Mucus

IOC for Immunological Infertility

ii)

CC + IUI

$\hookrightarrow$  6 Month

3 month CC alone

3 month CC + IUI

iii)

Mild Male factor  
Infertility

Semen  
Sample

Processing



Sample (highly  
fertile)

Sperm Swim up

technique & use Membrane  
Filter

iv) Disorders of Sexual Intercourse

$\hookrightarrow$  Vaginismus

Failure of erection

} Sexual  
dysfunction

v> Absence of Male partner (Popular in developed world).

GUG → Semen Sample Inserted  
O.P.D. Procedure ← { 0.5 ml (Max)  
Mixed with Liquid Media  
↓  
Put it in Uterus  
Preferably on the day of ovulation.

Pell's patient → to reach the site of fertilization & in  
to be injected 10 minute  
Position in Minimum  
of 10 min.

### Male Infertility Factors

MIC → defect in spermatogenesis

Semen Analysis → Best → Masturbation

Abstinence → 2d - 7day

Reach the Lab & in 60 min  
done after liquefaction

avg. time → 2-30 min

\* Wetter Parameter for Semen analysis 4

(151)

Lowermost value → for fertile

Volume  $> 1.5 \text{ mL}$

pH  $> 7.2$

Total sperm count  $\rightarrow > 39 \text{ million / ejaculate}$

(N)  $> 100 \text{ million / ejaculate}$

Sperm conc<sup>n</sup>  $\rightarrow > 15 \text{ million / mL}$

Total Motility  $\rightarrow > 40\%$

Progressive (Forward)  $\rightarrow > 32\%$

Morphology  $\rightarrow > 4\%$

Vitality  $\rightarrow > 58\%$

WBC Count  $\rightarrow < 1 \text{ million / mL}$

\* Aspermia  $\Rightarrow$  Absence of ejaculate

Azoospermia  $\Rightarrow$  Absence of sperm in ejaculate

Oligospermia  $\Rightarrow < 15 \text{ million / mL}$

Asthenospermia  $\Rightarrow$  (Abn) Motility

Telatospermia  $\Rightarrow$  (Abn) Morphology

Necrostermia  $\Rightarrow$  Dead Sperms



Globospermia - Sperm - Rounded head

↓  
Lack Acrosomal cap

\*

AZOOSPERMIA

↓ then 1<sup>st</sup>ly do

- Confirmation on a 2<sup>nd</sup> sample  
(1-4 week interval)

↓  
if azoospermia confirm

↓  
Do LH | FSH | Testosterone

if comes (N)

↓

Klas "Obstructive"  
Azoospermia" } ⇒ obstruction in  
Semen Pathway

→ if site of  
ejaculation block  
vas deferens

⇒ Scrotal USG

→ if site of  
ejaculation block  
on ED

(Volume ↓)

⇒ Trans-Rectal  
USG

↓  
dilated seminal vesicle

→ absent seminal vesicle

↓  
Cystic fibrosis (CFTR-gene)  
Analysis

if Non-obstructive case  
defective ⇒ (Testes) - M/c

↓

↑ LH | ↑ FSH | ↓ testosterone

↑ FSH | LH (N) | Testes (N)

↓

Sertoli cells affected  
(Leydig cells (N))

LH ↑ | Testosterone (N) / FSH (N)

↓

Partial AIS

↳ Testosterone (N)

FSH (Sertoli cell - inhibition)  
↳ LH reg

\* if Testes Involve  $\Rightarrow$  1<sup>o</sup> Hypogonadism (152)  
(Hypergonadotropic)

~~Testosterone~~  $\downarrow$  Testosterone

\* Non-obstructive Azoospermia

do for Non-obstructive Azoospermia cases  $\left\{ \begin{array}{l} \text{Microsurgical TESE (Testicular sperm extraction)} \\ \downarrow \\ \text{ICSI (Intracytoplasmic sperm injection)} \end{array} \right.$

\* if Sperm count (15 million/mL)  $\xrightarrow{\text{do}}$  IUI  
(5-15 million/mL)  $\xrightarrow{\text{do}}$  IVF  
(<5 million/mL)  $\xrightarrow{\text{do}}$  ICSI

\* Sperm conc<sup>n</sup> + Motility > Morphology (~~Most~~ Most imp. Parameter in - (N) sperm)

\* ART  $\Rightarrow$  Simplest form  $\Rightarrow$  IUI

All are ART except  $\Rightarrow$  ~~IUI~~

ZIFT

GIFT

ICSI

IVF

## GvF

- do for -
- i> Tubal factor infertility
  - ii> Male factor infertility
  - iii> Unexplained infertility



give 1st clomiphene citrate + gug X 3 cycles

for Superovulation; Not  
for ovulation induction

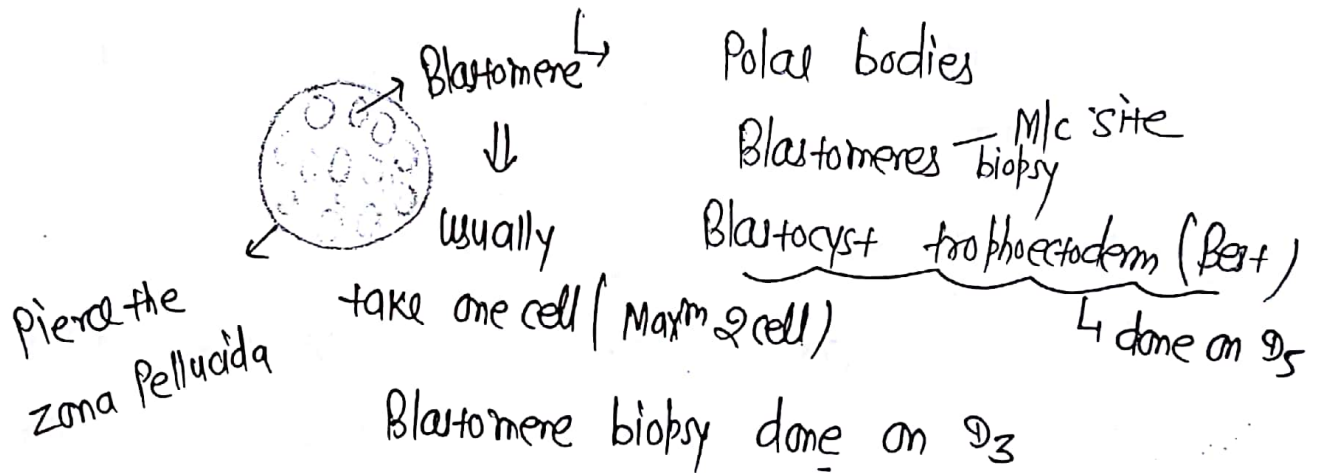
↓ if don't conceive

do gvf

iv> PGD (Pre-implantation Genetic diagnosis)



Site of Genetic Material taken



v> Premature ovarian failure

Part 8 of GvF → 1st ovulation induction

↳ by Gn: Gonadotropins



Follicular grow (Monitor growth)  
↓ by Number size; S.E.

(153)

↓  
Ovulation trigger by Gn. HCG

↓ 34-36 hr after

ovum pick-up (under USG guidance)

↓ on same day

GVF (test-tube Embryo formation)

↓

do Embryo transfer (M/c on D<sub>3</sub>).

↳ Never put one embryo; Min<sup>m</sup> - 2  
embryo puts at 2cm below fundus  
under USG guidance

### ENDOMETRIOSIS

- Presence of Endometrial glands & stroma outside the Uterus.

- M/c site ⇒ Ovary > Pouch of Douglas > <sup>leaf</sup> Posterior of broad Ligament  
Uterosacral Ligament > Fallopian tube

Occur at all sites except ⇒ Spleen.

- dependent on Estrogen for growth (ovarian steroids)

↳ so; disease of Reproductive age g/f (25-35yr)



- It is Rare in adolescent & Perimenopausal / Post Menopausal women

- In Pregnancy — Maxm time endometrium Regress

↳ b/c of Continuous - Progesterone Rich cond<sup>n</sup>  
↓  
decidualization

### PGI Theory of endometriosis

↳ Most accepted theory

↳ Retrograde Menstruation  
(Sampson's theory)

2<sup>nd</sup> Most accepted theory

↳ Coelomic Metaplasia

3<sup>rd</sup> ⇒ Immune Mediated theory

↓

Women w/ endometriosis have deficient cell Mediated / humoral immunity

4<sup>th</sup> ⇒ Genetic theory ⇒ K-ras

if one First degree Relative affected

↳ chance of endometriosis is 7 times more

5th  $\Rightarrow$  Lymphatic/ hematogenous

(154)

$\Downarrow$   
Umbilical Endometriosis

$\hookrightarrow$  Lymphatic explaining it very well

IOC  $\Rightarrow$  Diagnostic Laparoscopy

$\swarrow$   
confirm the diagnosis

$\searrow$   
Stage

$\downarrow$   
Biopsy (HPE)

$\hookrightarrow$  if altered coloured blood  $\oplus$

$\hookrightarrow$  Chocolate cyst  $\Rightarrow$  pr $\ddot{e}$  in ovary

$\downarrow$

Red flame lesion (New lesion)  
of endometriosis

$\hookrightarrow$  Powder burnt lesion (chronic lesion of endometriosis)

Minimal  $\rightarrow$  Superficial isolated implants

Mild  $\rightarrow$  Superficial + Multiple  
aggregates diameter  $< 5\text{cm}$

Moderate  $\rightarrow$  Superficial + deep lesion  
 $\hookrightarrow$  ( $> 5\text{mm}$  deep)

Severe  $\rightarrow$  if endometriosis distorts pelvic anatomy  
 $\hookrightarrow$  dense adhesion  
 $\hookrightarrow$  chocolate cyst

On USG  $\Rightarrow$  Chocolate cyst can be pickup

$\hookrightarrow$  Homogenous ground glass appearance

On MRI  $\Rightarrow$  Only pickup the chocolate cyst

$\hookrightarrow$  do in Adolescent girl comes  $\pm$  Endometriosis.  
 $\hookrightarrow$  "Mushroom gap sign" seen.

\* CA-125  $\Rightarrow$  Raised in Endometriosis

$\Downarrow$   
Normal  $< 35$

$\Downarrow$   
In Endometrioma  $\Rightarrow$  CA125  $> 100$   
(Rupture)

\*\*\*  
24/5/18

Presentation  $\Rightarrow$  Pain + Adnexal Mass + Infertility.  
(M/c)

$\hookrightarrow$  Menstrual complain — Menorrhagia

$\hookrightarrow$  Bowel/Bladder symptoms  $\oplus$

$\hookrightarrow$  Catamenial Hemo-thorax / Pneumo-thorax

$\hookrightarrow$  @ the time of Menses (May be endometriosis goes to Lung)

PAIN  $\Rightarrow$  M/c  $\Rightarrow$  Dysmenorrhoea  $>$  Chronic Pelvic Pain  $>$  Dyspareunia  $>$  Low Back Ache

$\hookrightarrow$  20 dysmenorrhoea

[ 10 dysmenorrhoea

d/t Progesterone withdrawal

20 dysmenorrhoea

d/t underlying disease process  
(Endometriosis)  
PID

<sup>1° dysmenorrhoea</sup>  
Spasmodic

Center (suprapubic)

begin Mostly on the 1st  
day of Menses

Improves  $\bar{c}$  blood flow  
clin 72 hrs - Relief

Rx  $\Rightarrow$  Very Responsive to  
NSAIDs

<sup>2° dysmenorrhoea</sup> 307  
Congestive (SS)

Localised (on one side)

PreMenstrual  
(Week before Menses)

doesn't improve  $\bar{c}$  flow;  
persist even Post Menstrual

Less Responsive to NSAIDs  
\* also has Dyspareunia;

$\downarrow$  seen in  
Rectovaginal Septum;  
deep endometriosis

Why Pain in endometriosis  $\Rightarrow$

Implants  $\rightarrow$  [Bleed]  $\rightarrow$  Inflammatory cells

$\downarrow$  Release

Inflammatory Mediators

Sign of Chronic Inflammation

cause of Pain

$\hookrightarrow$  healed by Fibrosis

$\hookrightarrow$  Cause Adhesion

$\hookrightarrow$  also cause Pain

\* Pain in - Implants

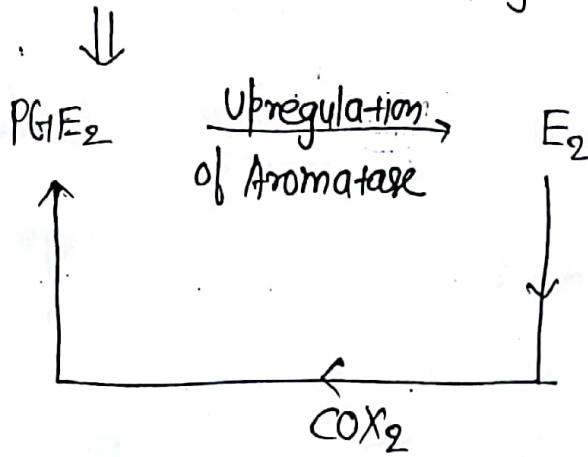
$\downarrow$

Neuromodulation ( $\uparrow$  Nerve Endings - Pain sensation)

$\hookrightarrow$  Cause by Estrogen,



\* Implants  $\rightarrow$  Estrogen is in Permanent Excess state



(N) Endometrium doesn't have excess Aromatase for Upregulation of Aromatase

$\Downarrow$   
[Eutopic]

(N) Endometrium has 17- $\beta$ OH dehydrogenase; which Metabolizes  $E_2$ .

\* What we do for Pain in Endometriosis  $\Rightarrow$

Pain (Suggestive of endometriosis)

Nature of Pain:

Minimal - Moderate



OCP's + NSAIDS Mediators



gt Suppress the ovary

if She wants to conceive then only NSAIDS given



If No Improvement

$\rightarrow$  Give Progesterone (do decidualisation)

Severe Pain



Give GnRH Agonist  
(given after diagnostic Laparoscopy)

Dienogest (Latest Progesterone)  
 $\hookrightarrow$  oral

oral MPA

DMPA (injection; works for 3 months)

Mirena (LNG - IUD)

(156)

if After Progesterone female  
is Not Responsible

↓  
Give GnRH Agonist. (given after confirmation of endometriosis)

↓  
by Laparoscopy

↓  
given in continuous manner

can also Give GnRH Antagonist.

↓ if Not Responsive

Give Danazol → highest Androgen S/E

Aromatase Inhibitor → it can produce hypoenstrogenic state

if given for more than six months ⇒ Result in Bone Loss;

So: given "ADD-BACK THERAPY" ⇒ Given NORETHINDRONE

↓  
to protect bone loss.

\* Surgical Management of Pain ⇒ i) Adhesiolysis;

ii) Fulguration of Gmplant;

iii) LUNA (Laparoscopic Uterosacral Nerve Ablation);

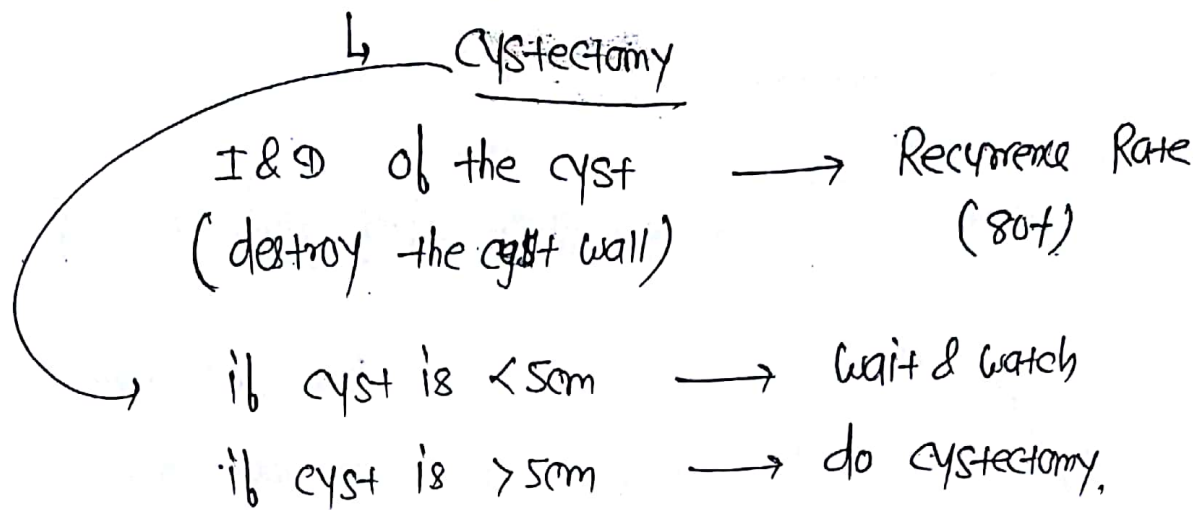
iv) Presacral Neurectomy

↳ Not effective;  
Not done Routinely.

v) Adrenal Mass

↳ Endometrioma ⇒ Not Responsive to Medical Mx at all

## \* TxOC for Endometrioma



## WID Hysterectomy (Last Resort)

↳ only in case of family is complete

## ADNEXAL MASS

Plv examination ⇒ findings who tells about endometriosis ⇒

- i) Tenderness in Pouch of Douglas;
- ii) tender Nodules on Uterosacral Ligament;
- iii) Fixed Retroverted uterus
- iv) Adnexal tenderness.
- v) Adnexal Mass

⇓

Ground glass appearance on USG

Infertility in  
Endometriosis

→ Main Reason ⇒ Ovarian (Not Anovulatory) (157)

{  
 - folliculogenesis ⇒ defective  
 - Genetic Material ⇒ Not good of ovm

In Moderate -  
Severe endometriosis

⇒ Main Reason ⇒ Ovarian + Tubal

In Minimal - Mild  
Endometriosis

⇒ Rx Clomiphene Citrate + Intrauterine  
insemination  
X 3 cycle  
(3 Months)

↓  
Superovulation

↓  
don't conceive

↳ do gvf

In Moderate - Severe  
Endometriosis

⇒ Rx GVF + Sx

↳ do Mainly; Surgery is Not  
- Necessary to do.

\* GnRH Agonists has No Role in improvement of Infertility  
in endometriosis

\* Endometrioma ⇒ if during the gvf, we find the chocolate  
cyst

⇓

wait & watch ← don't operate: b/c if operate, b/c of follicle damage;  
Infertility may seen.



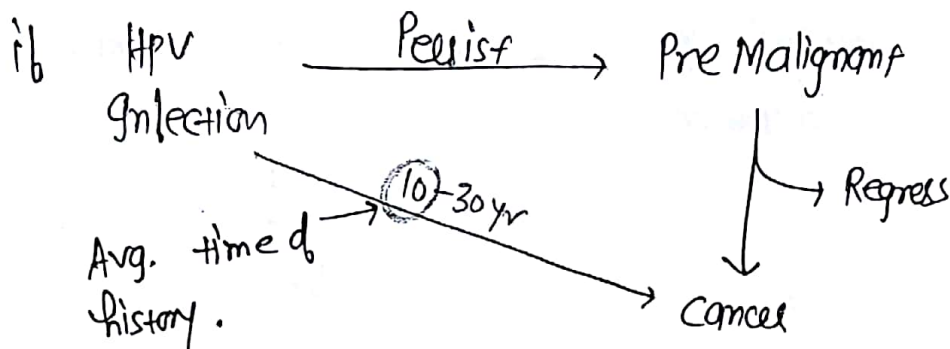
## \* MALIGNANCIES

\* M/c Cancer among Indian women  $\Rightarrow$  Breast Cancer  
2nd " " " "  $\Rightarrow$  cervical cancer

M/c cause of Mortality d/t Cancer in Indian women  $\Rightarrow$  "

## CERVICAL CANCER

Very commonly a/w HPV Infection (Most women clear this Infection)



If all History is given do "Universal Screening"

- Acc- to ACS/FIGO

Starts screening  $\Rightarrow$  21 yrs (irrespective of Sexual activity)  
In

How?  $\Rightarrow$  PAPS  $\rightarrow$  3 yearly in 21-25yr; everyone will turn out +ve; so: Not do.

Higher specificity  $\leftarrow$  PAPS + HPV (Co-test)  $\rightarrow$  5 yearly  $\rightarrow$  Higher sensitivity (Ideal  $\geq 30$ yr can do in  $\geq 25$ yr)

Stop  $\Rightarrow$  @ 65yr (provided the PAPS in last decade @)  
if Not Normal stop @ 75yr

WHO ProgrammeStart of  
Screening

⇒

begins @  $\geq 30$  yr3rd - 4th decade  
(30-49 yr)} more targeted  
Population Age  
Group.

WHO says "SEE &amp; TREAT".

How?? ⇒do single test

via HPV &gt; VIA &gt; PAPs

≡

(visual inspection  
w/ Acetic acid)

&gt; PAPs

↳ ideal  
↳ for confirmationBest Method ⇒ HPV + VIA

if HPV alone done ⇒ 5 yearly do

if VIA &amp; PAPs done ⇒ 3-5 yearly do

Treatment  
Protocol a/c to WHO

⇒

do cryotherapy (LEEP) &gt; conization

if Not

eligible goes for LEEP

\* Screening Methods → HPV testing (DNA testing)• For high-Risk viruses↳ do via Hybrid capture Technique

↳ expensive

• if HPV + PAPs → Co-test

• if do PAPs 1st then follow up by HPV ⇒ Reflex testing

## \* High Risk viruses

↳ HPV 16, 18, 31, 33, 35, 45, 52, 56, 58  
 ↓ ↓  
 70% of cervical cancer      30% of cervical cancer

\* Routinely in India we do ⇒ VIA (visual inspection w/ Acetic acid 5%)

↓  
Abnormal → White area (dysplastic cells ⊕)

↓  
 Aceto white area (stained test)

Unstained Area ⇒ Normal

VILI test

— Lugol's Iodine — 4-5%.

↳ Klein "Schiller's test"

↳ Brown ⊕ test — Normal cells w/ Glycogen  
 ↳ ⊕ in mature cells  
 ↳ Unstained (Abnormal test)  
 ↳ b/c of ⊖ of dysplastic cells (immature cells)

• PAPs Smear — Screening test (highly specificity)

↳ Taken by "Ayer's spatula & cytobrush".

↓  
 Pap smear is always taken from <sup>↳ taken secretion from endocervix secretion</sup> Bilid end of it & turn it by 360°.



Taken from Transformation Zone > Squamocolumnar Junction > Ectocervix of cervix,

\* Smear taken ON ONE SLIDE

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No Air drying do



Fixative — ?! <sup>AIIMS Nov/17</sup> 95% Ethyl alcohol + 5% ether

\* No Absolute Contraindication for PAP Smear; but if actively bleeding patient comes then tell her to come after bleeding stops.

\* PAP Smear is also k/w "Secretion Cytology"

\* BETHESDA CLASSIFICATION ⇒

① Normal Report

↳ tells about Report comes on PAP Smear.

② Reactive & Reparative changes (Healing Inflammatory changes);

③ Infection — Specify the organism

\* Organism lies inside endocx; tough to

- Catch in PAP Smear ⇒ Chlamydia  
Gonorrhoea

④ ASCUS — Atypical Squamous cells of Undetermined significance  
Asc<sup>us</sup> (Modified Bethesda classification)

⑤ LSIL — <sup>High Risk</sup> Low grade Squamous Intraepithelial Lesion

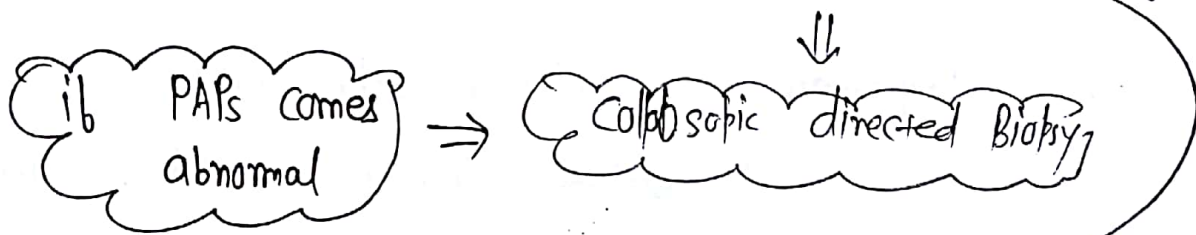
⑥ HSIL — High " "

⑦ Cancer



| * <u>Nucleus</u>    | <u>LSIL</u>                                | <u>HSIL</u> |
|---------------------|--------------------------------------------|-------------|
| <u>Cytoplasm</u>    | ↑                                          | ↑↑          |
| <u>No. of cells</u> | Less                                       | More        |
| <u>Granules</u>     | evenly distributed<br>(Granular chromatin) | clumps      |
| <u>Membrane</u>     | Shrivelled Membrane <del>Double</del>      |             |

Ideally → PAPS done → For Confirmation  
 ↓  
BIOPSY done  
 ( For looking Abn Area )  
 ↓  
 Use Colpo Scope → Focal Length = 30  
 Maxm Magnification = 30



We can see Ectocx  
 vaginal wall  
 vulva;  
 but we can't see Endocx

\* Abnormal Area from where Biopsy should be taken <sup>317</sup> ⇒

i) Irregular Surface contour

(160)

ii) Mucosa - Pale in colour

iii) In Acetic acid → white area seen

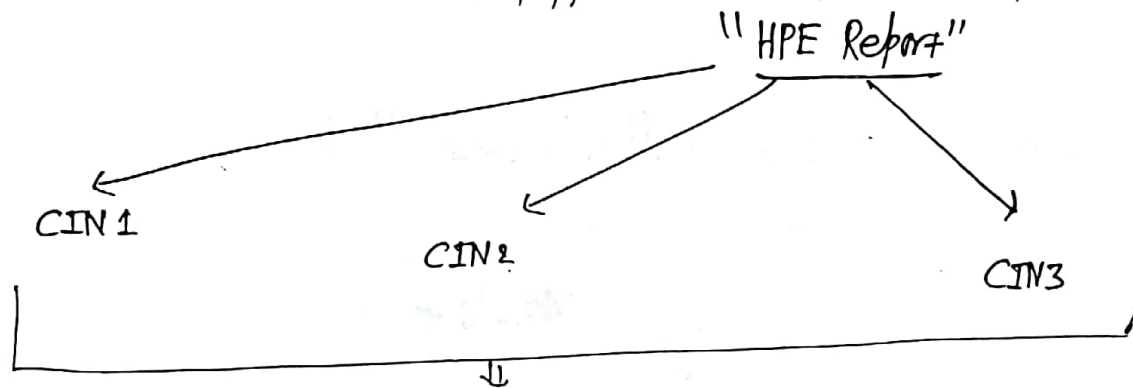
iv) In vascular pattern of

Reticular  
Mosaic  
Punctate

to visualise vascular pattern

clearly; we add Green filter to colposcope

\* Since it is Biopsy; so; Colposcope Report called as



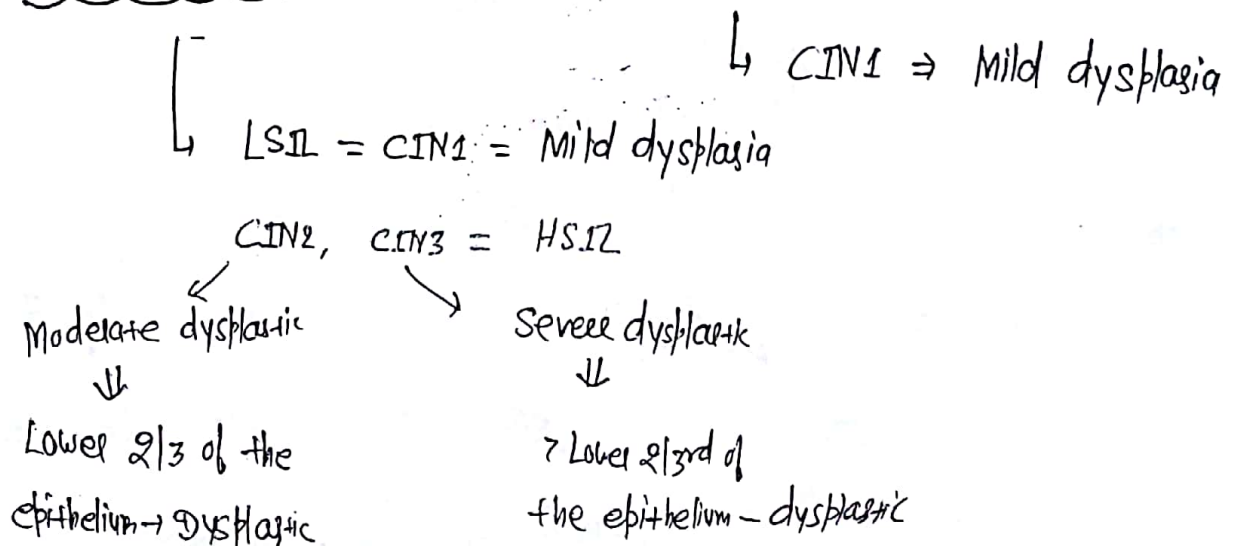
all are Pre Malignant cond<sup>n</sup> i.e all have basement

\* HPV cells affects basal & parabasal cells; so;

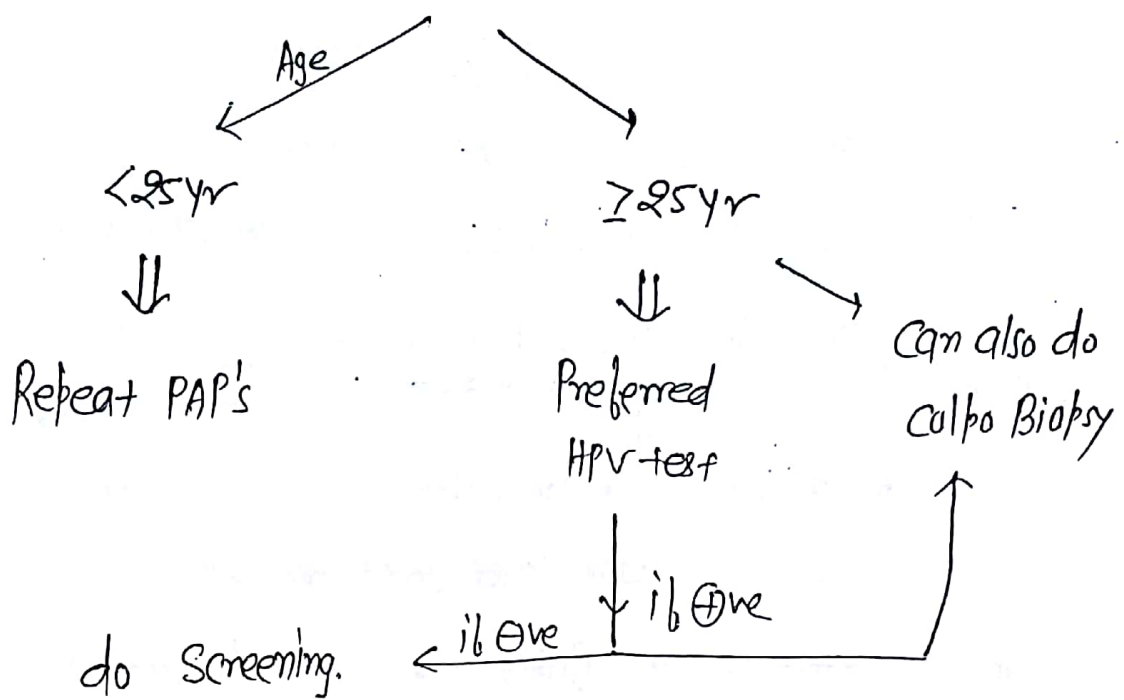
Membrane Intact.

Cancer starts from Lower part of epithelium

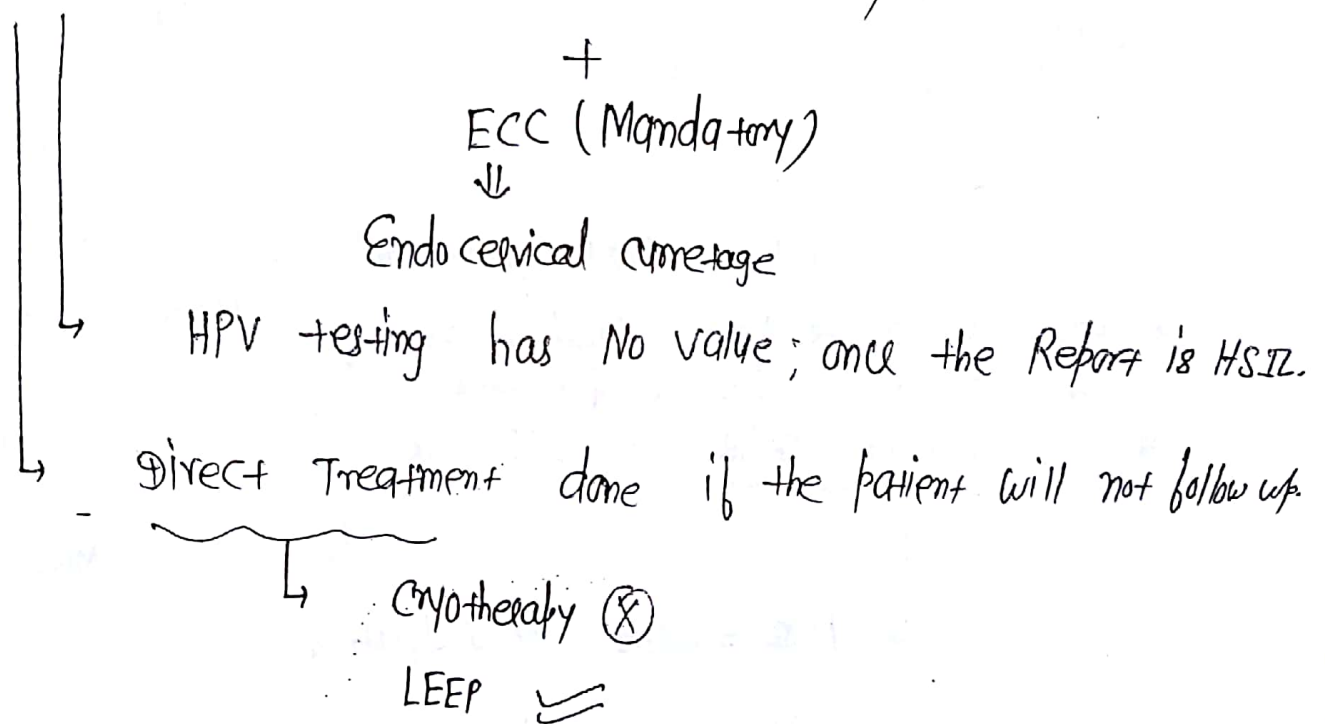
\* Dysplastic cells - all seen in Lower 1/3 of the epithelium



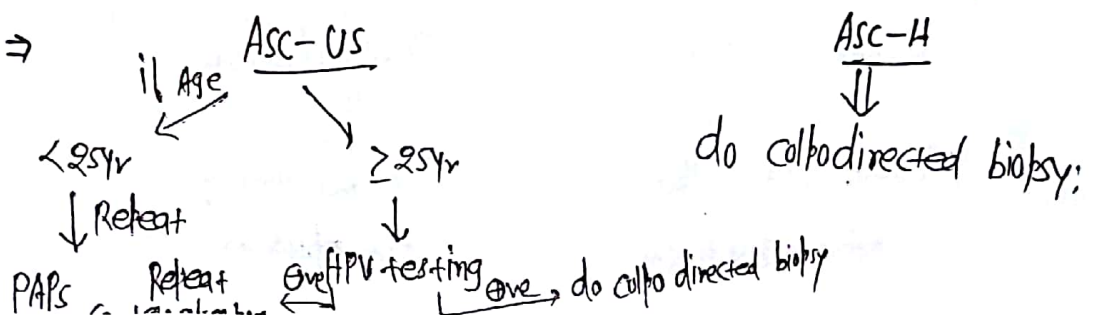
LSIL  $\Rightarrow$  if the case is of LSIL



HSIL  $\Rightarrow$  do colpodirected Biopsy



ASCUS  $\Rightarrow$



\* if Colpo Reports — Confirming the Lesion

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CIN-1



Cx 1-1.



Follow up



Co-test 1 yearly for 2 years

↳ Usual time of CIN1 to Regress



if Persistent for 2 years



Treat

CIN-2 → 5% (cancer) } Treat.  
CIN-3 → 12-40% }

\* How to Treat ⇒

Cryo-therapy > LEEP > Conization



Who is eligible — 22 criteria

all are known by visual inspection by acetic acid

- i) Entire Squamo columnar Junction visible
- ii) Entire lesion should be on Ectocervix
- iii) if lesion should occupy < 75% of Ectocervix

do CRYO ABLATION →  
↳ OPD procedure

apply cold gases (CO<sub>2</sub> / Nitrous oxide)  
↳ destroy the cells in tissue



## Mech<sup>n</sup> of cryoablation

↳ (Freeze - Thaw → Freeze)

do crystallization of Intracellular water



Causes Dessication

Pain Managed by Analgesics

Long term complication ⇒ Persistent Watery discharge

↳ bleeding is Not a complication of cryoablation.

\* Laser Ablation do → i) Depth of Lesion is More;  
→ ii) Lesion extends on the vaginal wall

\* LEEP = LLETZ →  
↓ ↓

Loop electro-  
Surgical excisi-  
onal procedure

Large loop  
excision of  
Transformation Zone

→ electric  
current

do cutting + Coagulation

\* Bleeding is Not the  
S/E of LEEP.

\*\*\*

No training Required;  
OPD Procedure!  
takes < 4min in completion of  
Procedure

\* Cryo-therapy doesn't give us any specimen; While LEEP give us  
specimen after procedure (Specimen of Transformation zone)

\* CONIZATION  $\Rightarrow$  Invasive ;  
O.T. procedure;

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Remove Tissue;

\* Indication of cone / C/I for LEEP  $\Rightarrow$

⊙  $\rightarrow$  Apex towards guide

$\rightarrow$  Cut from Transformation zone

Shallow  
cone

Deep  
cone

- ectocx  
endocx  
Junctional zone  
(Mucosa & stroma)

} given by cone

i) Unsatisfactory colposcopy

$\hookrightarrow$  entire Transformation zone is Not visible;

ii) When there is discrepancy b/w cytology & HPE

iii) if PAP's Smear is HSIL  $\rightarrow$  do Colpo. + ECC  
 $\hookrightarrow$  ⊕ve

iv) if there is Suspicion of Microinvasion

v) if Biopsy Report says  $\Rightarrow$  AdenoCa (Suspicion of endocx involvement).

Q.

38yr old Lady P<sub>3</sub>L<sub>3</sub> CIN3 ??

a) cryo

b) LEEP

c) Conization

d) Hysterectomy.

we need for follow up

→ only Indication to pre-invasive lesion

↳ In Recurrent CIN ;

• if Patient will not follow up ;

• if Suspicion of Adeno histology & family Complete

↓  
May be case of endometrial Ca;

so, do hysterectomy

• Other Pelvic pathologies which justify a hysterectomy.

## CANCER

R/F →  
For Cx  
Cancer

i) Early Age of 1<sup>st</sup> Intercourse

ii) " " " " 1<sup>st</sup> child birth;

iii) Multiparity

iv) Multiple sexual partner

v) Low Socioeconomic status

vi) STD

vii) Smoking → ↑ Sq cell ca  
⊗ Adeno ca.

VIII> Pre Invasive Lesion ⊕

ix OCP → ↑ Risk if use beyond or equal to 5yr

└ Nullify Risk if leave for ≥ 10yr

└ ↑ Related to Adenoca

x> family history

Early Menarche

Late Menopause

⊗ Not a R/F for Cx Cancer

Etiology of  
Cx cancer

⇒ HPV

└ also also cervix; vagina; vulval cancer;  
Penile; Anal; oral

High Risk HPV ⇒ Cause Cancer

Low Risk HPV ⇒ 6, 11 → Cause genital warts

⇓

Condylomata Acuminata

└ Laryngeal Papillomatosis

\* On HPE of HPV Infected cells

└ Koilocytes

└ Perinuclear halo

Cancer ⇒

E<sub>1</sub> E<sub>2</sub> E<sub>6</sub> E<sub>7</sub>

viral proteins

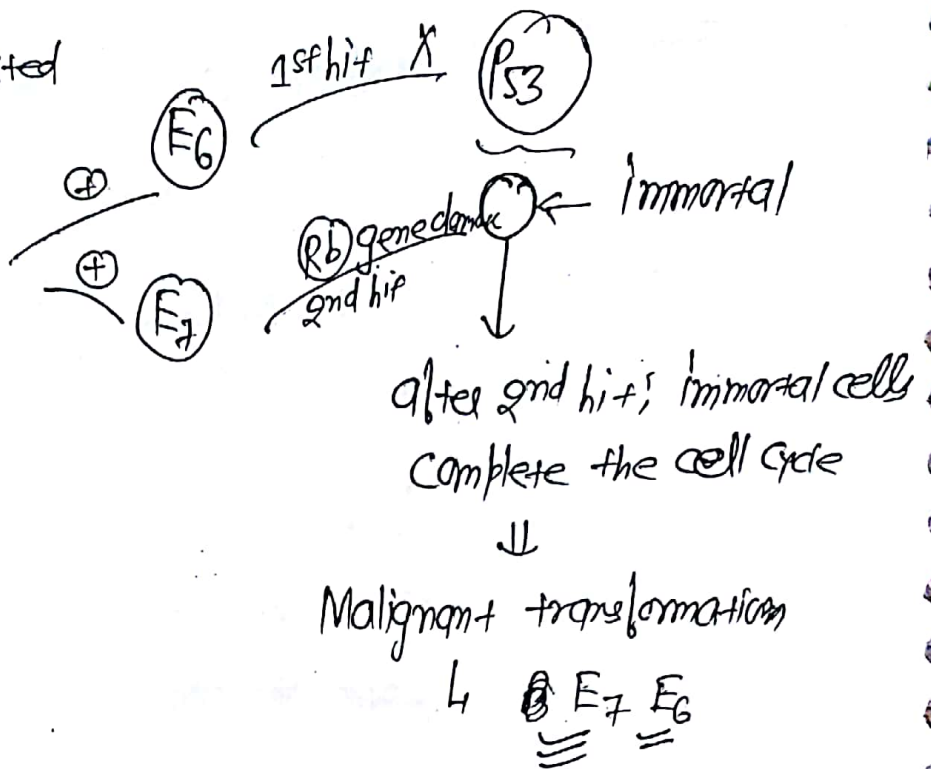
⊕ function ⊕



\* When Virus Integrated

in host genome

↓  
E<sub>1</sub>, E<sub>2</sub> altered



\* HPV vaccines

Gardasil

Quadrivalent

- Made from inactivated capsid protein

16, 18, 6, 11

- Latest FDA approved Gardasil-9 (Nonavalent)

16, 18, 6, 11, 31, 33, 45, 52, 58

- Given 0.5 ml i/m

- 0, 2, 6 Months

- Ideal age to give the vaccine ⇒ 11-12 yr  
can be given ⇒ 9 yr - 26 yr

- Girls/Boys both taken

CERVARIX

Bivalent

16, 18

given 0.5 ml i/m

0, 1, 6 Months

Girls taken

S/E of vaccine  $\Rightarrow$  Syncope attack

$\Downarrow$

So; there has to be observation time of 15min; then send her to home

(164)

\* Protection Rate - Quadrivalent Gardasil  $\Rightarrow$  70%  
Gardasil 9  $\Rightarrow$  95%

\* M/c virus a/w cancer  $\Rightarrow$  HPV16 = Squamous  
Most specific virus a/w cancer  $\Rightarrow$  HPV18 = Adeno

\* For cervical cancer  $\Rightarrow$

M/c Age  $\Rightarrow$  3rd-4th decade  
(Shows Bimodal Peak  $\rightarrow$  1st 3rd-4th decade  
 $\searrow$  2nd 5-6th decade)

M/c histology  $\Rightarrow$  Squamous cell ca = 69%  
Adenoca = 25%

Large cell Non-Keratinizing Squamous cell ca

M/c Route  $\Rightarrow$  Direct; Lymphatic

M/c Presentation  $\Rightarrow$  Irregular vaginal bleeding

1st Presentation  $\Rightarrow$

Most specific presentation  $\Rightarrow$  Post coital bleeding  
 $\downarrow$  Next  
Clinical examination

Clinical examination

↓ ⊕

do PAPs Smear\*

\* Persistent Postcoital bleeding  $\geq 6$  Months

↓

even with (N) PAPs

↓

do colpo Biopsy

Q 35yr P<sub>2</sub>L<sub>2</sub>  $\bar{c}$  Postcoital bleeding; o/E 2x2 cm growth on the Anterior lip of cervix  
do PUNCH Biopsy.

Q M/c site of distant Metastasis  $\Rightarrow$  Lungs  
In Cx cancer

M/c cause of death  $\Rightarrow$  Uremia (Renal failure)  
In Cx cancer

Risk of ovarian involvement  $\Rightarrow$   $< 1\%$   
In Cx cancer (Ovaries - Spared)

Most imp. Prognostic Marker  $\Rightarrow$  Stage  $>$  Lymph Node status  
In Cx cancer

# \* Staging for cervical cancer $\Rightarrow$

(165)

FIGO - clinical staging



Investigation should not be used to change stage of cancer:

USG  
CT  
MRI  
PET

(X) Not used.

Cystoscopy (to look bladder cavity);

↳ Part of Staging

EUA (Examination Under Anesthesia);

↳ Part of Staging

Stage 1

A<sub>1</sub> } - Micro - depth < 3mm; Horizontal Limit - < 7mm

A<sub>2</sub> } Invasion depth 3-5mm " - < 7mm

B<sub>1</sub> } → Macro - depth ≤ 4cm } & all Microscopic cond<sup>n</sup> above than A<sub>2</sub>.

B<sub>2</sub> } Invasion - depth > 4cm

Stage 2

Upper 2/3rd of vagina

A - Without Invasion of Parametrium

B - With Invasion of Parametrium

do MRI; but we can't  
↓ change the stage  
connective tissue

Clinically seen as  
obliteration of Fornices

Stage 3

A → Lower 1/3rd of vagina is Involved

B → Spread to Lateral Pelvic wall

(Ureter → hydroureter)

↳ seen by Gp/CT → approved to change stage  
to look for hydroureter.



Stage 4 : A → Spread to bladder and/or Rectum  
 B → distant spread  
 Inguinal L.N.

\* Early Stage Cancer ⇒ upto Stage IB<sub>1</sub>  
 ↳ Primary Tt ⇒ Surgery

Locally Advanced Cancer ⇒ ≥ IB<sub>2</sub>  
 ↳ Primary Tt ⇒ Chemo Radiation

Tt of Cancer Cervix ⇒

IA<sub>1</sub> → if Family is complete

↳ Hysterectomy

if Family is Not complete

(Type-I Hysterectomy / Simple Extrafascial hysterectomy)

↓  
 Fertility preserving conization.  
 (Therapeutic conization)



→ Cut Uterus; Cx & overlying fascia and Remove

IA<sub>2</sub> → if Family is complete

↳ Type 2 Hysterectomy + Pelvic LN  
 (Wertheim / Modified Radical) dissection

Cut midway b/w  
 Uterosacral Ligament  
 and/or cardinal  
 Ligament

Remove uterus;  
 Cx & half of Parametrium



↳ Lateral Pelvic wall

Parametrium

IA<sub>2</sub> - if Family is Not complete

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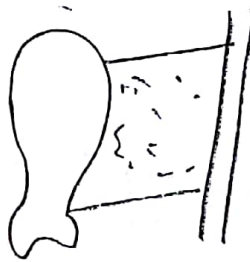
do Radical Trachelectomy



Remove Gx + Parametrium + Pelvic LN

Saves Uterus

IB<sub>1</sub> - if Family is complete



Type-3 Hysterectomy (Meigs/ Radical hysterectomy).

+

Pelvic L.N. dissection

+

Para-aortic LN Sampling.

↳ When we Remove all the Lymph Node

↳ Few L.N. Remove



Insertion on the  
Lateral Pelvic wall we  
cut the Cardinal/Uterosacral  
Ligaments

Type 1, 2, 3

Hysterectomy



only for Malignancy

(Piver Rutledge classification)

Q. Q. Cancer 1cm ; 4mm deep and 9mm H<sub>2</sub> Limit !! Stage →  
 In case if tumor is <sup>1B<sub>1</sub></sup> ≤ 2cm ⇒ type 2 Hysterectomy  
 (Wertheim's)

Mic Surgery in the Cx Cancer

\* 1-B<sub>1</sub> → Family is Not Complete

if ≤ 2cm ⇒ do Radical Trachelectomy.

### CHEMORADIATION

Stage ≥ 1B<sub>2</sub> ⇒



Chemo-therapy + Radio-therapy

(give concurrently)  
 Radiation sensitivity

Chemo Agent ⇒ cis-platin > SFU

In 2A1 (< 4cm)

↳ Type 3 hysterectomy



Upper 2/3rd of vagina

Removed (or half of vagina  
 Removed)

Not Recommendation; but we do.



\* Radio-therapy

→ distant

External beam RT

↓ close

(EBRT) - K<sub>125</sub> "Teletherapy"

Brachy-therapy (Intra cavity)

Mic used = Iridium

↳ 121

\* 1stly EBRT gives then follow up by Brachytherapy.

↓  
 Mic used = cesium

\* EBRT gives to Pelvis

(167)

↳ Dose  $\Rightarrow$  50 Gy in 5 weeks  
(5 fraction every weeks)

\* EBRT gives to Pelvis + Abdomen (Extended field RT)

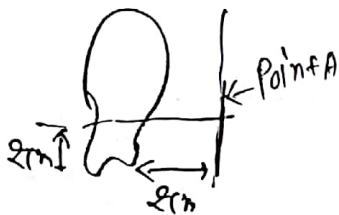
↳ Dose  $\Rightarrow$  30 Gy

\* Brachy-therapy gives to Pelvis

Point A

2cm above external  
OS

- 2cm Lateral to cervix

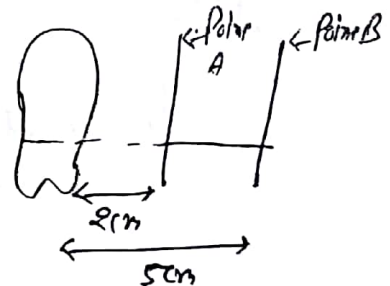


Corresponds to Paracervical  
Lymph Nodes; Ureters

Dose  $\Rightarrow$  8000 cGy

Point B

3cm Lateral to Point A at  
the same Level



Corresponds to Ureter

6000 cGy



Radiosensitive  $\Rightarrow$  Ovary > Rectum > Bladder > Vagina  
 order

L can tolerate 7000 cGy  
 L can tolerate 7500 cGy

L Most Radiosensitive  
 (can't tolerate Radiotherapy)  
 L for tolerant Mobilize it

## \* Adjuvant Chemoradiation

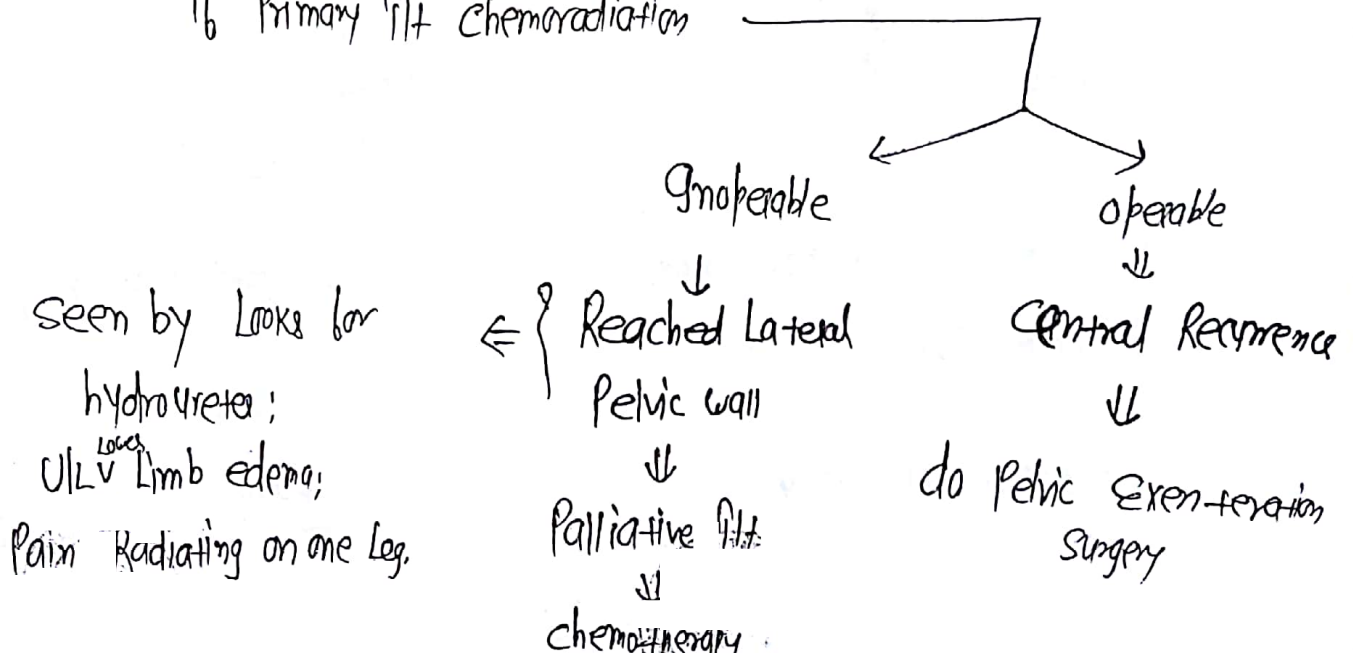
L give Post-operatively

Reports says  $\Rightarrow$  i) Lymph Node  $\oplus$   
 ii) Parametrium Invasion  $\oplus$   
 iii) Tumor Margins are  $\oplus$ ve

Q.Q. What to do in Recurrence of Cx cancer  $\Rightarrow$

Primary Rt if Sx  $\xrightarrow{\text{do}}$  chemoradiation

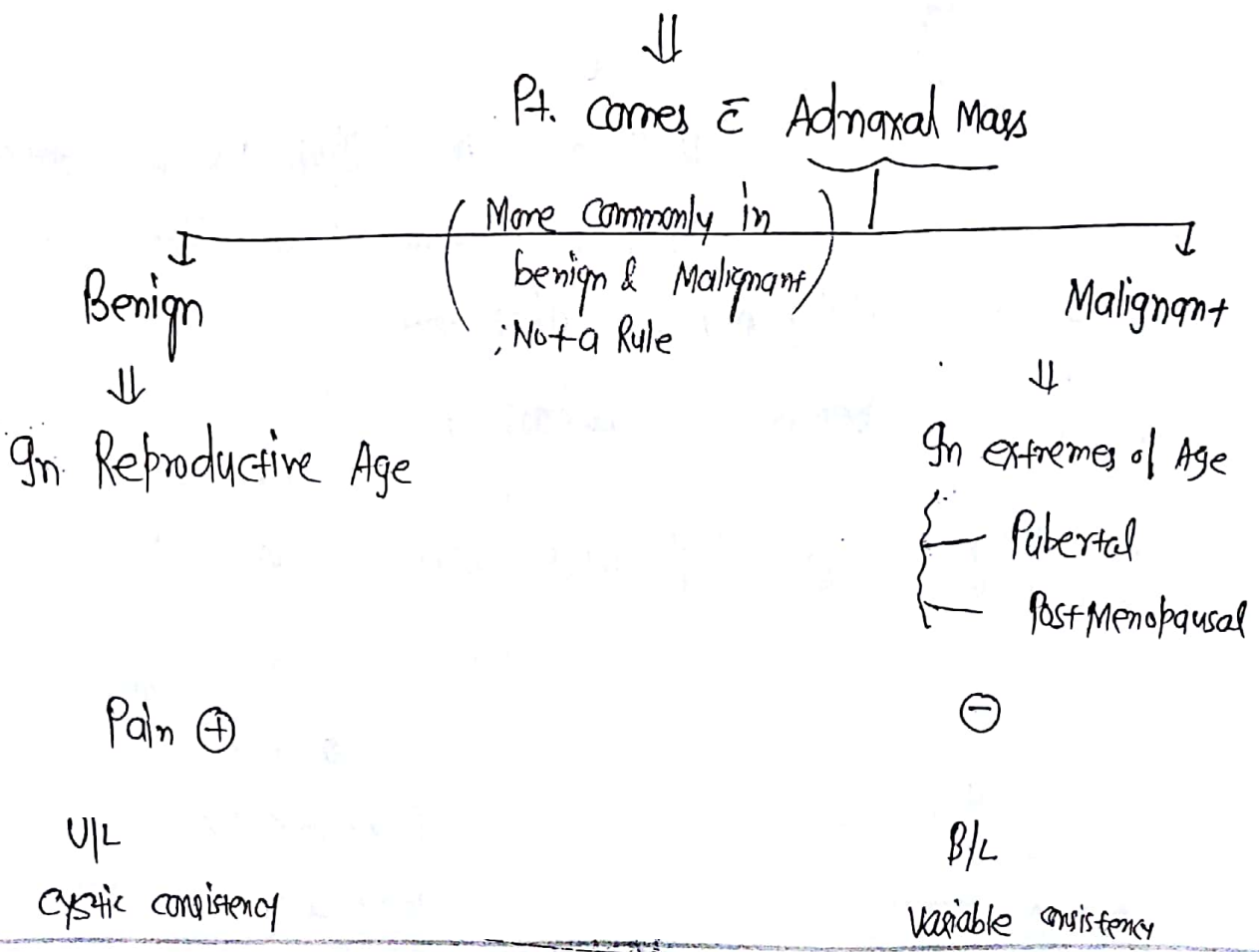
if Primary Rt chemoradiation



Q. Known case of Cancer cervix 3cm growth upper vagina  
 is involved  $\bar{c}$  Parametrium; CT - hydronephrosis; cystoscopy  
 bulba edema of bladder Mucosa (168)  
 Stage 3B.  
 doesn't Mean Invasion of bladder; i.e Lymphatics obstruction; so, Not change the stage

### OVARIAN CANCER

- Not very common like of cervical ca
- No specific sign & symptom
- Routinely Not screening for ovarian cancer



Benign  
tender

Malignant  
Non-tender

IOC for Adnexal Mass  $\Rightarrow$  TVS

{ U/L; Unilocular; Anechoic }  $\Rightarrow$  Benign cyst  
Most commonly

\* M/C cyst of the ovary  $\Rightarrow$  Follicular cyst\*

B/L, Solid component; septae;  $\nearrow$  ter vascularity; Ascites; Enlarged LN/  
(thick 2-3mm) / \ Matted Bowel Loop  
Ground Mass Septae



Malignant cyst. (Highly Risk of Malignancy)

Sx  $\Rightarrow$  Malignancy — Laparotomy

Benign — Laparoscopy

Indication

i) High Risk condition on USG

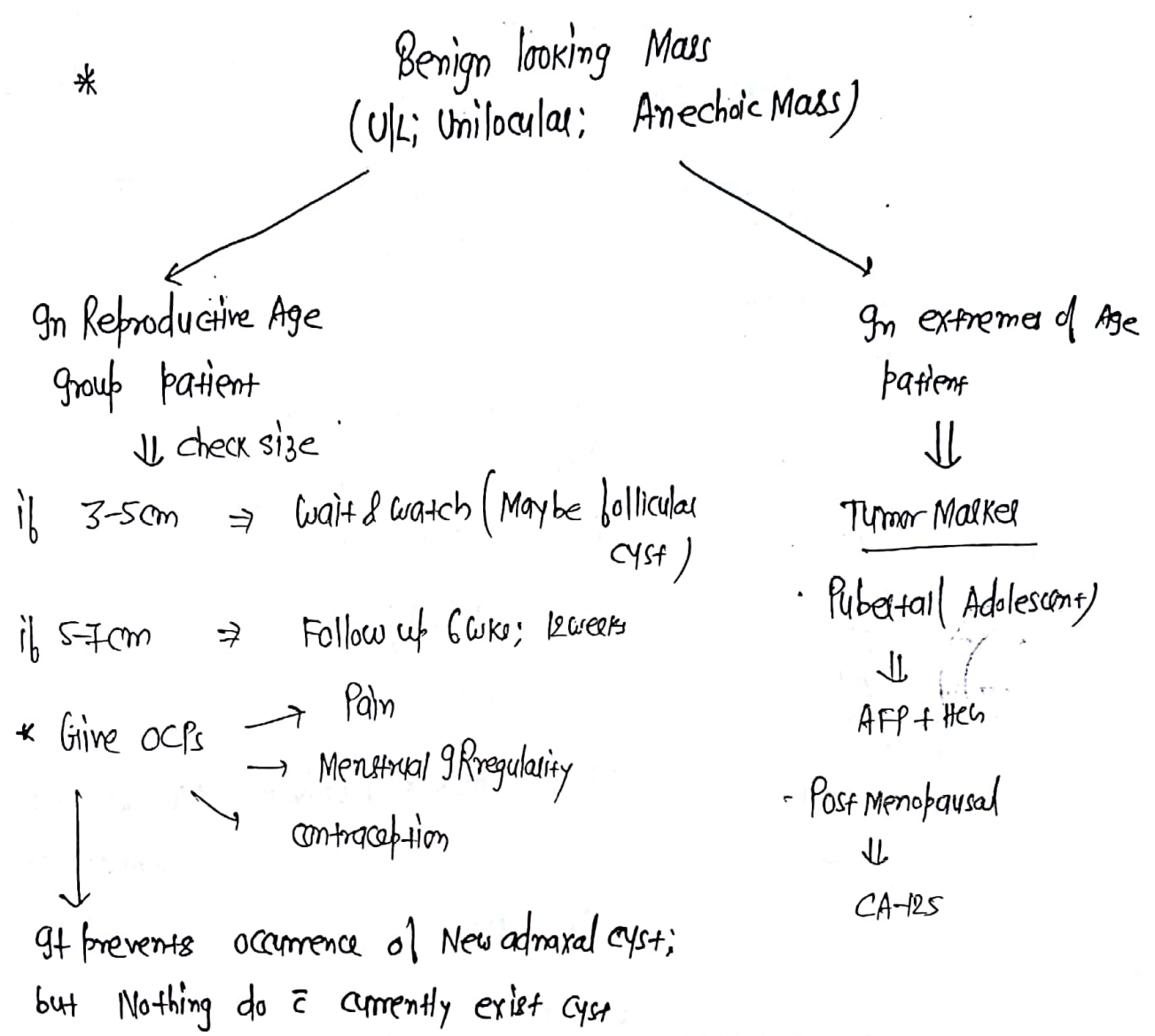
ii) Ovarian Mass > 7cm

Adnexal Mass > 10cm

iii) Raised CA125 (N value < 35)

↳ only in Post Menopausal age group.

iv Mass present as acute Abdomen <sup>d/t Rupture</sup>  
<sup>d/t Torsion</sup>



\* Adnexal Mass in a pregnant women =>

In 1st Trimester => wait & watch;  
 In 2nd Trimester => i) High Risk features  
 ii) >10cm (size)  
 iii) Acute Abdomen  
 Safest time for elective surgery.

cyst seen common in ♀ => i) Theca Lutein cyst → Hcg (Pregnancy Marker)  
 (Adnexal Mass) ↳ also in clomiphene citrate therapy



iii) Luteoma → In Pregnancy



• virilizing ovarian tumor

↳ In Mother

Less common - In fetus

- Spontaneously Regress after ♀.

More the ovarian cycle



More the Risk of ovarian tumor

More the estrogen



More the Risk of ovarian tumor

\* R/F for ovarian tumor

i) Early Menarche;

ii) Late Menopause;

iii) Infertility;

iv) obesity;

v) BRCA1  
2

HNPCC

vi) Smoking (only in Mucinous variety of ovarian tumor)

vii) PCOS

Protective for ovarian tumor



i) O.C.P

ii) Breast feeding;

iii) Only Anovulation (Not also Any disease)

iv) Salpingectomy  
Tubal Ligation  
Hysterectomy } Ascending Mitogens  
don't reach ovary

\* Fallopian tube ca ; ovarian ca & peritoneal carcinomatosis

↳ all have common origin ⇒ Fallopian tube

\* Types of ovarian tumor  $\Rightarrow$  i) Epithelial ; ii) Germ cells; iii) sex cord stromal

$\downarrow$  90%                       $\downarrow$  10%

### (A) Epithelial ovarian tumor (Features) -

- Serous - 75%

- Mucinous } 10% each

- Endometrioid

- Brenner

- Clear cells

Presentation Peak age - 60 yr.

(6th - 7th decade)

- B/L

- Non-specific sign & symptom

↳ Gravitational bowel Sx

- Late stage

- High Mortality Rate

- Sporadic - 90%

$\downarrow$   
So, only 10% tumor are familial.

Can tend to occur  
a decade earlier  
(50yr)

Gene Involved  $\Rightarrow$  BRCA1 <sup>\*\*1</sup>/<sub>545</sub>  
BRCA 2 - 25  
HNPCC - 15%

\* If First degree Relatives affected  $\rightarrow$  Risk 1x by 3 times.

Q. If patient is K/clo Genetic Mutation  $\rightarrow$

Go for Prophylactically Sx

$\downarrow$   
Go for BSO (Beyond 35yr Risk 1x every year)

Bso (B/L Salpingo-oophorectomy)

↓  
done @ 35yr or as soon as family complete

↓  
This surgery protects against ovarian cancer  
also protects from Breast cancer by 50%.

2nd Line approach

⇒ if Patient doesn't want to go for Bso

↓  
Give OCP's + Screening - High Risk

↓  
High Risk { i) Strong family Breast/Ovarian ca  
ii) K/c/o Genetic Mutation

Screening ⇒ TVS + CA125

Start @ 35yr 6 Monthly / 12 Monthly

Ovarian Tumor

⇒ M/c ovarian tumor = Serous cyst Adenoma  
M/c ovarian ca = Serous cyst Adenocarcinoma

\* Mucinous ovarian tumor ⇒ i) Decade earlier

ii) Grow to Large Size (20cm)

iii) Diagnosed early

iv) Better Prognosis

v) UEL (B/L in 10% cases)

vi) CEA (Tumor Marker)

CA125  
↓ Tumor Marker  
for Serous tumor

VII> Pseudomyxoma Peritonei (171)

(Loculated mucinous collections in Abdomen)

→ M/C Seen in tumor of Appendix; Not ovary.

\* Endometrioid ovarian ca - high Risk for coexistent "Endometrial ca".

↳ 10% of total  
Epithelial  
↳ U/L in Nature

\* clear cell ovarian ca - highest association with Endometriosis

↓  
also a/w Endometrioid ovarian ca.

↳ HobNail cells ⊕ (○)  
↳ HPE findings.

\* Brenner's - U/L; Benign; solid lesion

↳ on HPE - Bladder Like epithelium - Transitional epithelium.

⑧ GERM CELL TUMOR → 5-8% of all ovarian tumor

• Teratoma → Mature cystic (Dermoid)  
Teratoma

↳ Immature

• Dysgerminoma



- Embryonal cell ca
- EST (Endodermal Sinus tumor) / Yolk sac tumor
- Choriocarcinoma
- Mixed tumor

M/c Germ cell tumor of ovary  $\Rightarrow$  Dermoid

M/c Germ cell cancer of ovary  $\Rightarrow$  Immature  $\succ$  Dysgerminoma  
Teratoma

\* Feature  $\Rightarrow$  i) U/L (Unilateral);  
of GCT Which GCT has highest Risk of Bilaterality  $\Rightarrow$   
Dysgerminoma  $\succ$  Dermoid  
(15%) (10%)

ii) Seen in younger girls (10-30yr)  
 $\downarrow$

What % of ovarian tumor in this  $\Rightarrow$  70%.  
Age gp. are Germ cell tumor

iii) Non-specific sign & symptom + (A) Precocious Puberty  
(B) acute abdomen

iv) Pick up in early stage

v) Good Prognosis

vi) Conservative.

b/c GCT Release hcg  
 $\downarrow$   
& Submit similar to LH/FSH  
EST / Yolk sac tumor  
 $\hookrightarrow$  Max + Rapid growth of tumor

\* Dysgerminoma  $\Rightarrow$  TUMOR MARKER  
 $\Rightarrow$  LDH (HCG + PLAP)  
 (Tumor Marker)  
 ↓  
 Flethy; Lobulated &  
 Tan in colour. Not secrete AFP.

(172)

\* Endodermal Sinus tumor  $\Rightarrow$  AFP (LDH)  
 (Tumor Marker)  
 ↓  
 Not secrete HCG.

\* Choriocarcinoma  $\Rightarrow$  HCG

\* Embryonal  $\Rightarrow$  HCG + AFP

\* Dermoid  $\Rightarrow$  No Tumor Marker

↓  
 Rarely HCG secrete

↓  
 Rokitansky Protuberance / Tip of Goeborg sign

↓  
 White Area Inside cyst (black) in USG.

\* M/c ovarian tumor in Reproductive Age  $\Rightarrow$  Dermoid.  
 " " In ♀  $\Rightarrow$  Dermoid  
 " Cancer  $\Rightarrow$  dysgerminoma

\* Dermoid  $\Rightarrow$  Benign

Risk of Malignancy (0.2-2%)

type

site

Sq cell Ca

Rokitansky  
Protuberance

↓  
 M/c ovarian tumor to go in torsion,

\* Germ cell cancer  $\bar{c}$  Best prognosis  $\Rightarrow$  Dysgerminoma  
" " worst prognosis  $\Rightarrow$  EST / Yolk sac tumor

\* Only ovarian tumor  $\bar{c}$  is Radiosensitive

$\Downarrow$   
Dysgerminoma (Moderately Radiosensitive)

### © Sex cord stromal tumor (3+)

- Granulosa cell tumor
- Sertoli-Leydig cell tumor
- Leydig cell tumor
- Thecoma
- Fibroma (stromal tumor of ovary)

Features  $\Rightarrow$  , U/L : occur in all age gp / Peak incidence in Perimenopausal women;

- Non specific sign/symptom,
- AUB  $\longrightarrow$  Estrogen  
or  
virilization  $\longrightarrow$  Testosterone
- early stage pick up
- don't show any Lymph Node Metastasis
- Best prognosis:
- Granulosa cell tumor  $\longrightarrow$  Inhibin  
 $\hookrightarrow$  Secrete estrogen  $\rightarrow$  Risk of endometrial ca.





### Stage 3


A — 1  $\Rightarrow$  One Retroperitoneal Lymph Node  
2  $\Rightarrow$  Microscopic extra pelvic peritoneal spread

B — Macroscopic  $\leq 2\text{cm}$  extra pelvic peritoneal spread

C — Macroscopic  $> 2\text{cm}$  extra pelvic peritoneal spread  
 $\hookrightarrow$  if there is involvement of capsule of Liver/spleen

### Stage 4

A  $\rightarrow$  Malignant pleural effusion

B  $\rightarrow$   Parenchyma of the abdominal organ  
distant spread  
Inguinal Lymph Node

Stage 1/2  $\Rightarrow$  early Stage  
Ca

Stage 3/4  $\Rightarrow$  Advanced stage  
Carcinoma

\* For early diagnosis  
of ovarian Ca  $\Rightarrow$  TVS  
or

K/C/O ovarian Ca  $\Rightarrow$  Best  $\Rightarrow$  CT-Scan  
 $\hookrightarrow$  can't do staging CT-Scan

\* On Follow up; if CA 125  $\uparrow$   
 $\hookrightarrow$  it Means Recurrence happen  
 $\downarrow$   
do PET scan

# Staging Laparotomy $\Rightarrow$ Steps

(174)

i) Midline vertical incision

$\hookrightarrow$  Never give Pfannenstiel incision or transverse incision; Rarely Paramedian incision given



ii) Ascites  $\rightarrow$  Sample taken  $\rightarrow$  Cytology for malignant cells  
OR

Saline wash  $\rightarrow$  50-100ml  $\rightarrow$  Cytology for malignant cells

iii) Inspection & Palpation of all abdominal organs

iv) Random Peritoneal biopsies



Paracolic gutters

Pouch of Douglas

Surface of diaphragm  $\rightarrow$  Scrapping (acceptable)

v) TAH  $\pm$  BSO (Pan hysterectomy)

$\downarrow$   
type 1 hysterectomy

vi) infracolic omentectomy.

vii) Pelvic & Paraaortic LN Sampling

viii) Closure

\* In Stage 3/4  $\Rightarrow$  Primary T/t  $\Rightarrow$  Surgery  
 $\Downarrow$   
Debulking Sx

\* Post-operative  $\Rightarrow$  Chemotherapy.

\* Conservative Sx for  $\Rightarrow$  U/L Salpingo-oophorectomy  
ovarian cancer

$\hookrightarrow$  eg  $\Rightarrow$  Germ cell tumor

(U/L; Younger age patient)

• Stage IA (if fertility is desired).

• Borderline epithelial ovarian tumor

$\hookrightarrow$  epithelial ovarian potential  $\bar{c}$  Low  
Malignant potential

(Stromal Invasion - Absent)

Very good prognosis

U/L

decade earlier

## Post-operative chemotherapy

347

(175)

↳ Epithelial  $\Rightarrow$  All stages except 1A & 1B grades  
cell ca Need Post-op. chemotherapy,

Chemotherapeutic  
Agent  $\Rightarrow$

Carboplatin + Paclitaxel } 6 cycles  
 $\downarrow$   
Cisplatin + Paclitaxel  
given i/v + Peritoneal

Germ cell ca }  $\Rightarrow$  All Stages Need chemo. except  $\Rightarrow$   
sex cord ca }  
 $\downarrow$   
Dysgerminoma stages  
II

No Need of  
Chemotherapy

Only Advanced stages Need chemotherapy.

Chemo.  
Agent  $\Rightarrow$

BEP (Bleomycin, Etoposide, cisplatin).

## ENDOMETRIAL CANCER

Etiopathogenesis  $\Rightarrow$   $\uparrow$  Estrogen (unopposed)

$\uparrow$  Menstrual cycles

• RIF  $\Rightarrow$

Early Menarche;

Late Menopause;

PCOS;



iv Infertility

v obesity

vi HTN

vii Diabetes

} → Endo ca (Carpus Cancer Syndrome)

viii Tamoxifen

→ highest - 70+

ix BRCA1, 2, HNPCC → Lynch Syndrome

↳ also ovarian ca; Not of Breast Cancer

x HRT (only Estrogen)

Protective  
of Endometrial ca

• Smoking

• OCP (↓ Risk by 60+)

(↓ Risk of ovarian ca by 50+)

• Exercise

• Green tea

Histology

⇒

Type 1

Endometrioid

Most common in 80+ cases

Estrogen Responsive

Pregnant

Good Prognosis

Type 2

Papillary; serous; clear cells

In 20+ cases

Non Responsive

(X)

Poor prognosis (Worst prognosis clear cells)

Type I  
Gene association  $\Rightarrow$  PTEN  $\leftarrow$  Gatekeeper Gene  
KRAS

Microsatellite deletions

Type II  
p53

(176)

High grade Serous show p53 Mutation

Low grade Serous show KRAS Mutation

Peli Menopause  
Early Menopause

Later More Age More type II

Obese Lady

thin Lady,

\* Pre Invasive Lesion  $\Rightarrow$   
which changes into the  
Endometrial Ca

Endometrial hyperplasia

$\Downarrow$   
on HPE by taking endometrial Biopsy.

Klau "cystic glandular hyperplasia" } Glands & stroma both proliferate

Simple hyperplasia

Clout Atypia  $\rightarrow$  1+

Complex II

"  $\rightarrow$  3+

Simple  $\bar{c}$  Atypia

$\rightarrow$  8+

Complex  $\bar{c}$  Atypia

$\rightarrow$  29+

Glandular Proliferation is Much more than ~~Complex~~ <sup>Stromal</sup> prolifer

$\Downarrow$   
Back to Back arrangement of gland.

## Hyperplasia c/out Atypia

↳ given Progesterone 1<sup>st</sup>ly

M/c  $\Rightarrow$  MPA X 6 Months

(daily)



Repeat Biopsy

also we DMPA

Mirena

## Hyperplasia c Atypia

Reconfirm  $\leftarrow$  L do Hysterectomy (TOC)

(Frozen section)

↳ Intraoperatively send & get a  
Immediate Report & Reconfirm

$\Downarrow$  if Not possible

do Endometrial sampling (Optional)

(FC + Hysteroscopy)

↳ Functional curettage

2nd Line  $\Rightarrow$  if wants Preserving the Uterus

Reconfirm on Repeat Endometrial sampling  
(FC + hysteroscopy)

$\downarrow$   
2ndly Progesterone (Prefer - Megestrol Acetate X 6 months)  
Other MPA  
Mirena

\* Simple hyperplasia  
Cystic glandular hyperplasia

→ Metrorrhica  
Hemorrhagica

351

(177)

Age  $\Rightarrow$  40-45yr

8 weeks of Amenorrhoea  $\Rightarrow$  History of bleeding

(Anovulatory cycles  
b/c Unopposed Estrogen)

$\Downarrow$   
Painless

Endometrium  
on HPE  $\Rightarrow$

Absence of secretory pattern

Swiss cheese appearance

Ovary  $\Rightarrow$

Cyst

Ca  $\Rightarrow$

1%.

TOC  $\Rightarrow$

Progestelone

\* Peak age for  
endometrial cancer

$\Rightarrow$  60yr (5-7th decade)

M/c presentation / 1st presentation = Irregular vaginal bleeding

Most specific presentation = Post Menopausal bleeding (PMB)

% of PMB have endometrial Ca = 10%

M/c cause  $\Rightarrow$  Senile endometrial Atrophy  
(Atrophic endometrium)

M/c cause of PMB in India  $\Rightarrow$  Ca cervix



\* M/c Cause of Pyometra  $\Rightarrow$  Endometrial ca  
In India  $\Rightarrow$  cervical ca

\* M/c Route for endometrial ca  $\Rightarrow$  Direct spread  
" " ovarian ca  $\Rightarrow$  Tumor Exfoliation

\* No Routine Screening done for endometrial ca

$\downarrow$   
if pt. is K/c/o "HNPC" - do Routine Screening

$\hookrightarrow$  age  $\geq 35$  yr

do functional curettage (FC)

6 monthly / 12 monthly.

\* Any women comes  $\bar{c}$  Post Menopausal bleeding

$\hookrightarrow$  Rule out Endometrial ca

\* if women  $\geq 45$  yr  $\subseteq$  AUB

$\hookrightarrow$  Rule out Endometrial ca

Next Step

do TVS (see endometrial thickness)

$\Downarrow$   
ACC to ACOG if thickness  $\geq 4$  mm  $\rightarrow$  high Risk for cancer  
" American Radiologist "  $\geq 5$  mm

AIIMS May 18

16 endometrial thickness  $< 4\text{mm}$  /  $< 5\text{mm}$   
 (ACOG) (American Rodology) (178)  
 ↳ No Test In Risk of Cancer

\* IOC  $\Rightarrow$  Endometrial Biopsy  
 \* TVS do Polyp to Rule out other pathologies like fibroid;  
 O.P.D Procedure; Aspiration do (Endometrial Aspiration cytology)  
 ↓  
 we use "KARMAN'S CANNULA"  
 Other device "PIPELLE DEVICE"  
 "VATIRA ASPIRATOR"

\* Gold Standard technique to Rule out endometrial ca  
 ↓  
 Most Invasive  
 $\Rightarrow$  Functional curettage + Hysteroscopy  
 May (D&C) use synonym  $\bar{c}$  Functional curettage  
 O.T. + Anesthesia Needed  
 ↳ 1<sup>st</sup> ly do cervical curettage - (Endocervical curettage)  
 ↓ then  
 dilate Internals  
 ↓ then  
 Endometrial curettage  
 ↳ 1<sup>st</sup> ly do endometrial cells comes in cervix.

## Condition where Functional cure stage done

- i) Endometrial biopsy — Benign ; while Symptom Persistent
- ii) Endometrial biopsy — No Endometrial cells seen
- iii) Hyperplasia  $\bar{c}$  Atypia  
(preserve the Uterus)
- iv) Cervix Stenosed

\* Should we do PAP Smears in Post Menopausal bleeding  $\Rightarrow$  Yes.

Staging  $\rightarrow$  FIGO  $\rightarrow$  Surgical Staging

Stage 1  $\left\{ \begin{array}{l} A - \text{Only Endometrium or } < 50\% \text{ of Myometrium} \\ B - > 50\% \text{ of Myometrium} \end{array} \right.$   
Intraoperatively Cut open the Uterus

Stage 2 — cervical spread

$\Downarrow$   
only if cervical stroma is involved

Stage 3  $\left\{ \begin{array}{l} A \rightarrow \text{Cervical spread to Serosa/Adnexa} \\ B \rightarrow \text{'' Vagina/Parametrium} \\ C_1 \rightarrow \text{+ve Pelvic LN} \\ C_2 \rightarrow \text{+ve Para-aortic LN} \end{array} \right.$

Stagey A  $\Rightarrow$  Spread to Bladder and/or Rectum (179)  
 B  $\Rightarrow$  Distant spread / Inguinal L.N.

### Staging Laparotomy

$\hookrightarrow$  Omentectomy is Not done Routinely

$\hookrightarrow$  for type 2 Endometrial ca.

do TAH  $\pm$  BSO

$\hookrightarrow$  type 1 — Stage 2 — Type 3 hysterectomy  
 $\Downarrow$   
 Cervical Spread

- Pelvic & Paraaortic LN sampling  $\Rightarrow$  Type 2 cancers  
 L.N. dissection

- Stage 3/4  $\Rightarrow$  Advance stage Ca

$\hookrightarrow$  Primary Rx  $\Rightarrow$  Debulking surgery

### Post-operative

#### Low Risk

- all three
- a) Endometrioid
- b) Grade 1
- c) only endometrium

#### Intermediate Risk

In b/w Low & high  
 Risk

#### High Risk

- a) Stage 3/4 disease  
 OR
- b) type 2 cancer



Tlt of Low Risk

Need No Post-operative Tlt;  
do Follow up

Tlt of Intermediate Risk →

Pelvic Radiotherapy

Tlt of high Risk →

Chemotherapy + Radiotherapy

↓  
(Cisplatin + Paclitaxel)

PROLAPSE → Recent classification  
↳ POP-Q classification  
↳ Pelvic organ prolapse  
quantification  
↓  
Reference → Hymen.

\* Delancey Level of Support

Level I — Uterosacral Ligament + Cardinal Ligament  
↓  
Weak → Uterocervical descent

[ earlier classified as

1<sup>st</sup> degree → descent above vaginal opening

2<sup>nd</sup> degree → Uterus descent @ the level of vaginal opening

3<sup>rd</sup> degree → Uterus descent outside the vagina

Procidentia → Uterus completely outside the vagina

↳ Fundus is also outside

Apical Prolapse

Enterocoele → Prolapse of GUT into Pouch of Douglas.

Vault Prolapse (apex of vagina)

(Post hysterectomy)

Uterocervical descent

Level 2 ⇒ Paravaginal tissue & their attachment  
 basia covering Levator Ani (180)

↳ Weak  $\xrightarrow{\text{causes}}$  Cystocele (upper 2/3rd Anterior wall)  
 ↳ M/c type of Prolapse

Level 3 ⇒ Perineal body & Muscle attach to it

↳ Weakness  $\xrightarrow{\text{causes}}$  Rectocele (Post. vaginal wall) L ⇒ LEVATOR ANI MUSCLE;  
 E ⇒ EXTERNAL ANAL SPHINCTER

\* Prolapse is disease of older Age.

RIF of Prolapse ⇒ i) No. of deliveries & process deliveries  
 (Prolonged Labour)

Instrument delivery  
 Episiotomy "  
 Perineal tear  
 obstructed Labour

ii) Old Age (d/t loss of estrogen)

\* Younger age women also have Prolapse of uterus by →  
 May

- i) Connective tissue d/o.
- ii) Congenital elongation of cx
- iii) Spinal cord injury

\* Smoking ↑ Risk of Prolapse

\* Intra abdominal Pressure ↑ ⇒ ↑ Risk of Prolapse

↳ Chronic cough; Ascites.

Case I ⇒ Old woman P<sub>3</sub>L<sub>3</sub>; 3rd degree Uterocervical descent  
+ cystocele + Rectocele

Uterocervical descent → do Vaginal hysterectomy

Sequence of cutting  
& clamping the  
structure in TAH



Reverse the sequence  
of vaginal hysterectomy

Sequence of cutting & clamping of sequence

Firstly Uterosacral Ligament



Cardinal Ligament



Uterine Artery



Utero ovarian Pedicle

\* Sequence of cutting & clamping the structure in TAH & BSO ⇒

Uterosacral  
Ligament



cardinal  
Ligament



Uterine  
Artery → Infundibular  
Ligament



Utero ovarian  
Pedicle



\* Usually we prefer vaginal hysterectomy; but  
it is difficult in — Uterine size > 12wk

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(181)

Obese

Pelvic Adhesions.

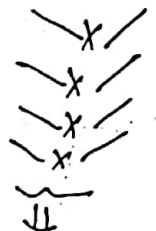
### \* Urinary Tract Injuries

↳ Laproscopic Sx > Abdominal > Vaginal  
hysterectomy hysterectomy  
↳ highest Risk for Urinary tract injuries

↳ In Benign cond<sup>n</sup>

↳ Seen in hysterectomy for Malignancies  
(Wertheim's hysterectomy)

\* Rx of Cystocele → Anterior colporrhaphy  
(Vagina)



Interrupted Suture

Rx of Rectocele → Posterior colporrhaphy

Strengthen the Levator Ani Muscle



VH + Ant. colpo

+ Posterior colpo/epineorrhaphy

VH + Pelvic Floor Repair

+ Enterocoele Sx

Ward Mayo Sx\*\*

do prophylaxis; also do  
for vault + Prolapse

McCall's Culdoplasty  
↳ vaginal Repair

Other Surgeries for Enterocoele ⇒ Moschowitz Repair  
Halban's Repair  
Both are Abdominal Repair; Not used Now a day

Support the vaginal vault =

Uterosacral Ligament in McCall's Culdoplasty.

2nd case

Younger - Reproductive  
Age group

+ Prolapse

(Uterocervical descent)

TOC ⇒ Sling's Surgery

↳ can't do hysterectomy

↳ Modified Shirodkar's abdominal Surgeries

Anterior sling

(If we tie's at anterior aspect of Gsthamus)

\* Pyramid Sx

↳ Autologous (tendon fascia lata)  
- and - Allogeneic external oblique muscle

Posterior Sling

(If we tied at posterior aspect of Gsthamus)

\* Khamis's Sling.

## Khamma's sling

(182)

↳ one end at Posterior aspect of Isthmus;  
Other end Anterior Superior Iliac spine.

## Shriodkar Sling ⇒ Posterior sling

↳ Merselene tape used  
on the left hand side first pass through  
Psoas Muscle (Form Psoas hook) then goes to sacrum

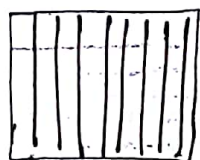


to prevent compression & obstruction  
of sigmoid colon / Rectum.

## VikRud's sling ⇒ Composite Sling (Anterior + Posterior sling)

Abdomino cervicopexy ⇒ For putting Mesh we have to  
clear Pre-Sacral Area; technically  
difficult; but Results are bet.  
higher complication

Mesh



## \* Non Sling Surgery ⇒ Manchester Repair

(Klas" Fothergill's Surgery)  
↳ done in Reproductive age women who completed child bearing.

- also done in congenital Cx elongation  
UCL (Uterocervical Length) Estimation

- Process  $\Rightarrow$   
of Manchester  
Repair

$\downarrow$   
D & C (to prevent the complication of  
Cervical stenosis)

$\downarrow$   
Cervical Amputation

$\downarrow$

Reattach Cardinal Ligament anteriorly

$\downarrow$

Cover the Cervix  $\rightarrow$   $\bar{c}$  vaginal Mucosa

Cystocele done

Rectocele Repair done

if the UCL is  $\uparrow$ ; don't do Sling operation; do  
Manchester Repair

UCL = Uterocervical Length

(N) UCL Length  $\Rightarrow$  6 to 8 cm.

\* Shriodkar Modification of Manchester

$\downarrow$   
L Can be done even if women want  
to conceive.

$\downarrow$   
All steps are same in it except there is No cervical  
Amputation in it



3rd case

old woman + Co-morbidities

(183)

PAC  
(Pre-Anesthetic  
Check-up)

bit for short Sx.

~~Latzko colporrhaphy~~

↓

done for vesico-vaginal  
Fistula.Rx ⇒ LeFort's colpocleisis

Denude Mucosa - Anterior vaginal wall  
Denude Mucosa - Posterior vaginal wall

Means scrape  
Mucosa from Anterior  
& Posterior vaginal  
wall.

1stly we Rule out Cx Cancer & Endometrial  
Cancer

&amp; Patient can't be Sexually active

• Done Under Local Anesthesia.

{ colpocleisis ⇒ closure of vagina }

4th caseCo-Morbidities + old woman↳ PAC ⇒ Unfit for short Sx.

Rx ⇒

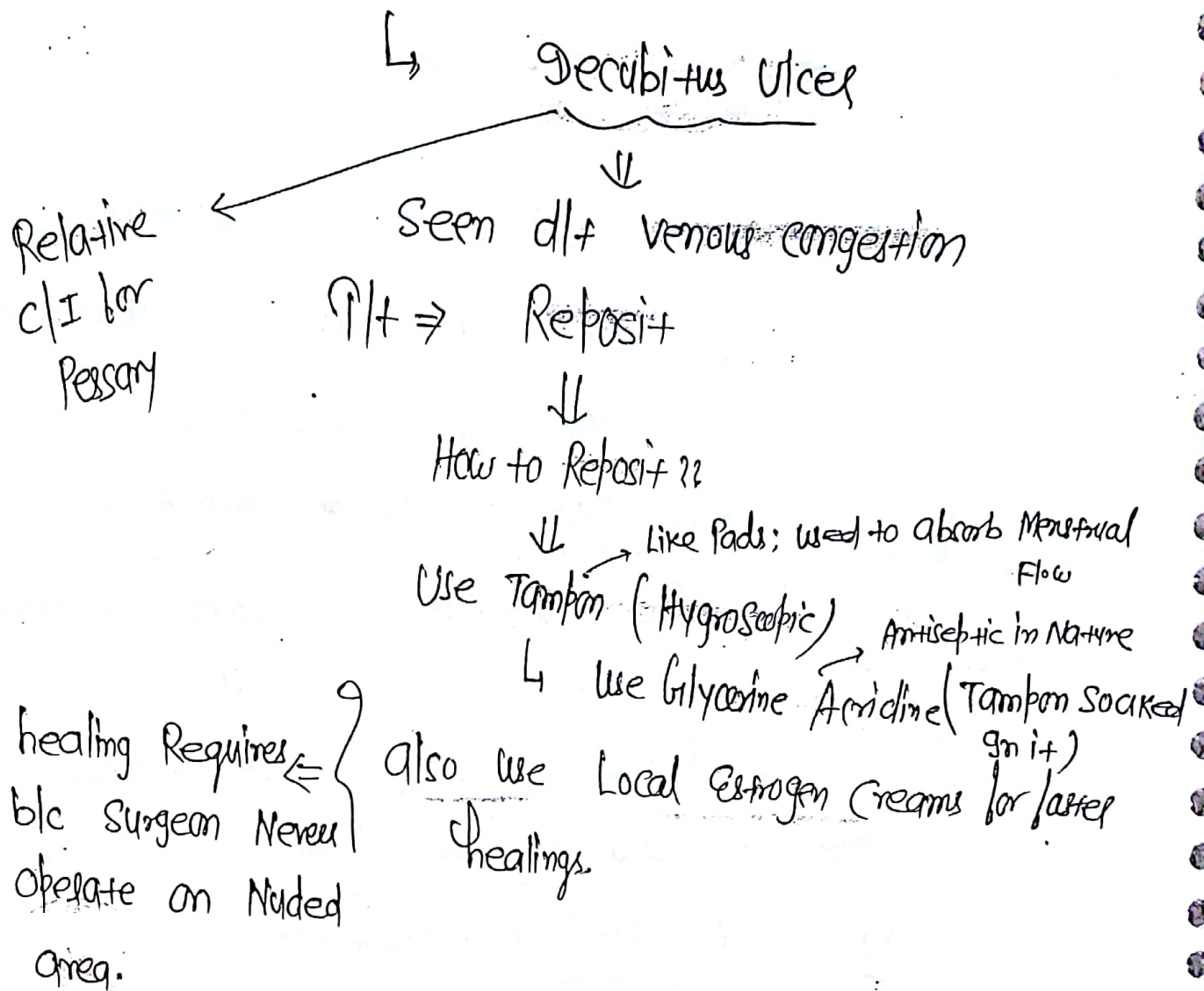
Pessary→ space occupying device  
- Insert into vaginaGellhorn  
PessaryDoughnut  
Pessary

\* Early ♀ Prolapse - also Indication of Pessary  
↳ Remove - 16-18 weeks.

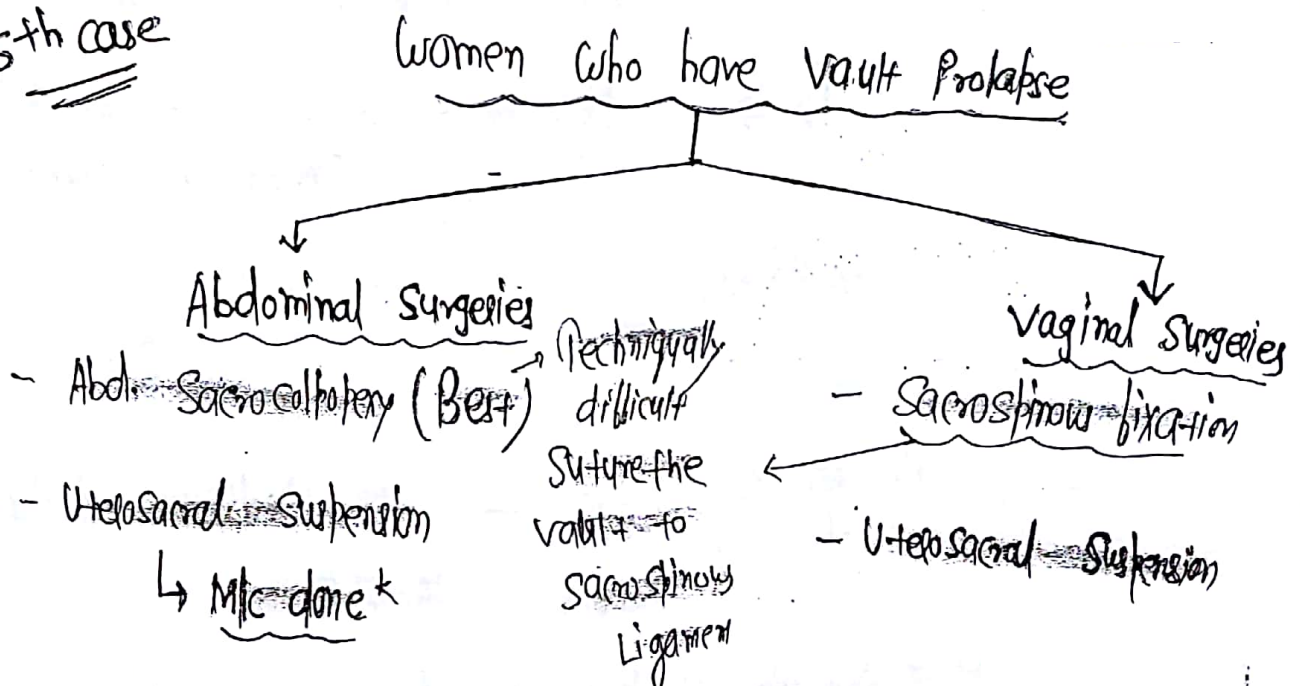
\* Other Indication of Pessary - Puerperium



# \* Ulcer on Prolapse part

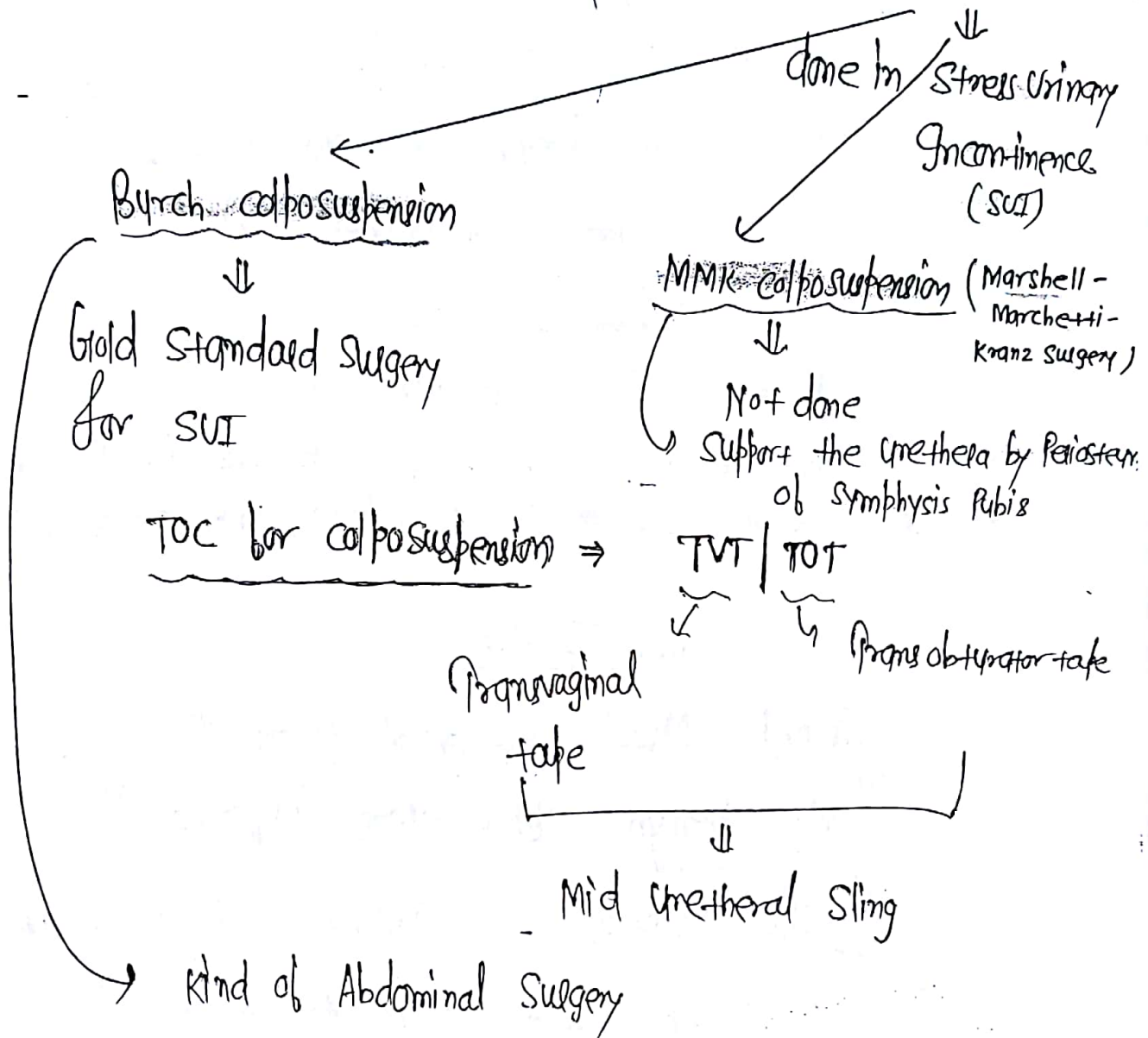


5th case



\* LeFort Colpocleisis can also done in vault prolapse (184)  
 ↳ In short time Sx.

\* all all Sx of vault Prolapse except Colposuspension:



- Support the proximal urethra by Cooper's Ligament.

- ↳ They are as good as Burch colposuspension
- ↳ Day-care Sx;
- ↳ Lesser complication;
- ↳ vaginal Surgeries;

\* Trans vaginal tape  $\Rightarrow$  Repubic Surgery

Trans obturator tape  $\Rightarrow$  Not Repubic Surgery

$\hookrightarrow$  In it we enter the Space of Retzius

$\Downarrow$

So; More complication than TOT,  $\therefore$  So;  
We prefer TOT Now a days.

\* Pt  $\bar{c}$  Vault prolapse + SUI??

$\Downarrow$

Abdominal Sacrocolpopexy

Rx  $\Rightarrow$  Burch colposuspension.

\* 1st Line Mx of SUI  $\Rightarrow$  Kegel's exercise i.e Pelvic floor exercise

\* Drug Rx of SUI  $\Rightarrow$  DULOXITENE (only drug Rx of SUI).

### FIBROID

- Smooth Muscle tumor of Uterus <sup>99</sup>

- M/c benign Pelvic tumor in females. <sup>99</sup>

- a/w Estrogen;  $\therefore$  Seen in Reproductive Age  
in  $> 35$ yr women; it is More common.

- In Postmenopausal women  $\rightarrow$  Regression Seen.

- In Pregnancy — Most of the fibroid don't enlarge



Types → grow within myometrial wall, <sup>kk</sup> Intramural (Mlc) ⇒ 75%

(185)

Subserosal  
grow outward towards the peritoneal surface  
Submucosal  
grow toward into different types the uterine cavity,  
Most fibroids to begin as Intramural & later converted

Hysteroscopic appearance (Wansteke's classification).

→ Type 0 → Completely Intracavitary

Type 1 → >50% intracavitary i.e. <50% in Myometrium

Type 2 → <50% intracavitary i.e. >50% in Myometrium  
↳ can't be removed hysteroscopically

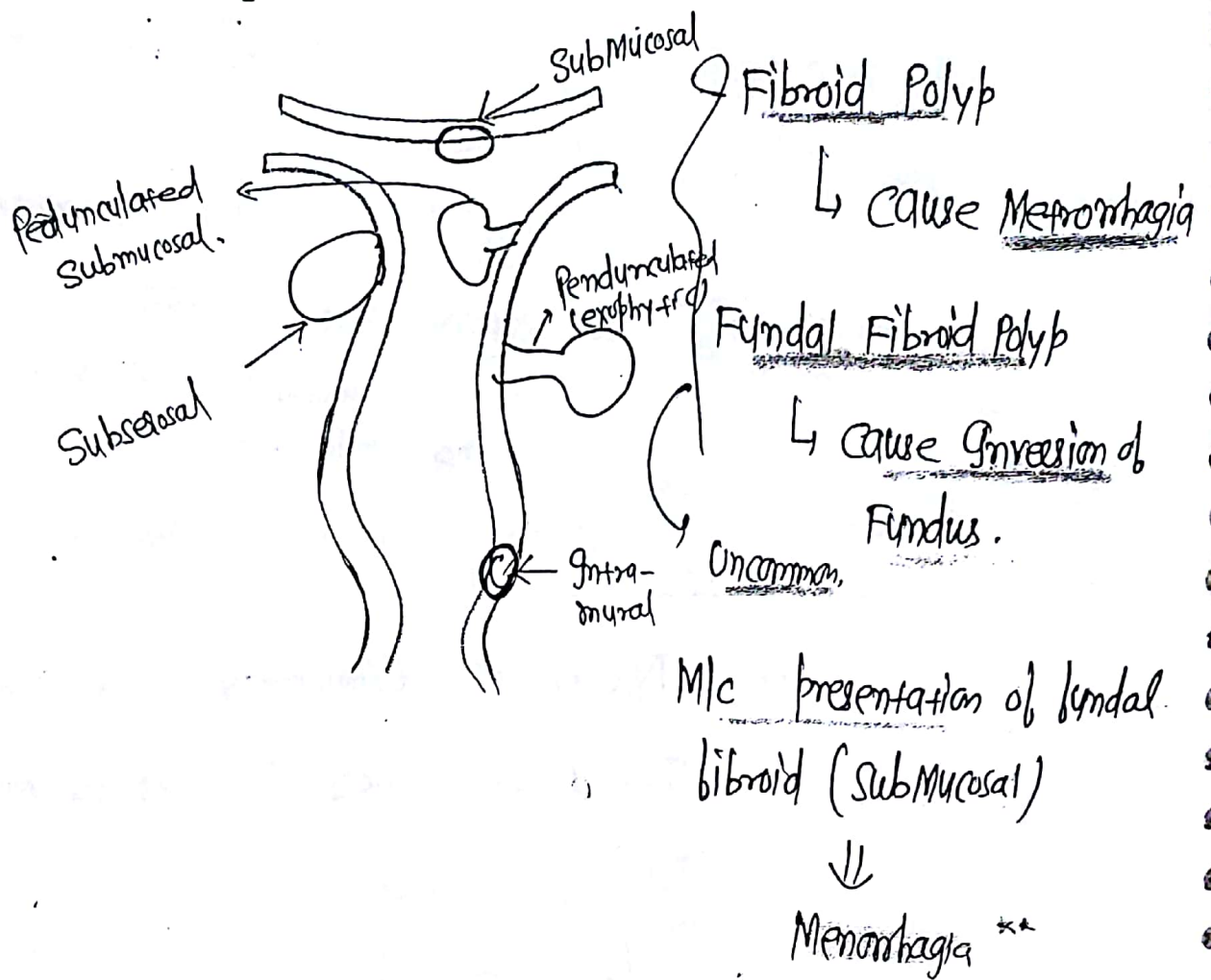
Mlc presentation ⇒ Asymptomatic

Mlc Symptom ⇒ ① Bleeding → Menorrhagia

↓  
Cycles — Regular  
Flow — ↑↑↑  
Mlc cause by which fibroid  
↓  
Submucosal

While Subserosal fibroid Not a/w bleeding





② Pain → it undergoes - degenerations

Torsion - Pedunculated Subserosa

Muc degeneration ⇒ Hyaline \*\*\*

Least Common " ⇒ Sarcomatous \*\*

↓  
Rare Malignant change

↳ < 0.5%

↳ Postmenopausal fibroid enlarge & pain.

\* Womb Stone - Subserosal fibroid Stone; (186)  
 ↳ Old bladder stone  $\downarrow$  calcification  
 ↳ it is eccentric  $\rightarrow$  Differential diagnosis

\* Red degeneration  $\Rightarrow$  Seen in ♀  
 $\downarrow$   
 • Stained Salmon Pink<sup>a</sup> or Red  
 • Fishy odor.<sup>a</sup>  
 • Histologically: evidence of thrombosis in some vessels.<sup>a</sup>  
 - M/C in 2nd Trimester.  
 - presenting as acute Abdomen (Pain + Nausea; vomiting  $\pm$  Fever)  
 - Mx - Conservative  
 N.P.O + i/v fluids + analgesic  
 $\rightarrow$  aseptic cond<sup>n</sup>  $\Rightarrow$  No Role of Antibiotic \*\*  
 ↳ Fever is only Reactionary

\* In Fibroid  $\Rightarrow$  dysmenorrhoea  $\oplus$ ; but Not a primary complaint.

\* Fibroid doesn't cause dyspareunia

Fibroid May show Pressure symptom

↳ Ant Wall Fibroid  $\Rightarrow$  Urinary symptom  
Post wall Fibroid  $\Rightarrow$  Bowel symptom \*\*

\* Ant. wall fibroid cause  $\Rightarrow$  ↑ Frequency of Micturition  
 Post. " " "  $\Rightarrow$  Urinary Retention

\* BROAD LIGAMENT FIBROID

False



Subserosal uter. fibroid  
 outgrows into the  
 Layers of Broad Ligament

True



denovo b/w the Layer  
 of broad Ligament

Lateral to the  
 Fibroid



Uterus



Medial to the  
 Fibroid.

\* Fibroid also pref. Infertility  
Recurrent @ Loss

SubMucosal Fibroid  
only \*\*

\* Pregnancy complication of Fibroid

apart from RPL &  
Red degeneration

Abortion  
 Pre-term Labour  
 Malpresentation  
 dysfunctional Labour  
 PPH



IOC  $\Rightarrow$  USG (Hypoechoic)

↳ Well-circumscribed Masses;  
Pseudocapsule

Small Submucosal fibroid May miss in USG

↓

Best Ix  $\Rightarrow$  Hysteroscopy

2nd Best  $\Rightarrow$  SIS (Saline Infusion Sonography)

\* Don't do MRI  $\Rightarrow$  Routinely

↓

do in pre-op. cond<sup>n</sup> to know

No.

size

Location

\* Diff

Fibroid

• Smooth Muscle  
tumor

• > 35yr age

• Menorrhagia

• Non tender

• Irregular growth  
(Lumpy Bumpy growth)\*

• Grows upto uterus sized  
20 weeks

Adenomyosis

Endometrial glands & stroma  
Infiltrate Myometrium

40-45yr age

Menorrhagia + dysmenorrhea

Tender (Half hen size)

Symmetrical Growth  
(Globular)\*

10-12 weeks



## Fibroid

GOC  $\Rightarrow$

USG

alternate  
dark & light  
band

### \* Degenerations/ Secondary changes in Fibroid

$\rightarrow$  Mnemonic

- 4 Avoid  $\rightarrow$  Atrophy;
- Red  $\rightarrow$  Red degeneration;
- Hot  $\rightarrow$  Hyaline degeneration;
- Fatty  $\rightarrow$  Fatty degeneration or calcification;
- Meat of  $\rightarrow$  Myxomatous degeneration;
- chicken  $\rightarrow$  Cystic degeneration.

don't tell about  
where endometrium starts & myometrium ends

For confirming  $\Rightarrow$  (HPE) - Postoperatively  
Adenomyosis

See depth of these  
endometrial glands in myometrium

At least 1 HPF deep  
2.5mm deep to the junctional zone

TOC  $\Rightarrow$  Hysterectomy\*

## Adenomyosis

MRI

On USG we see  $\Rightarrow$

Salt & pepper appearance:

venation blind "

Myometrial cyst

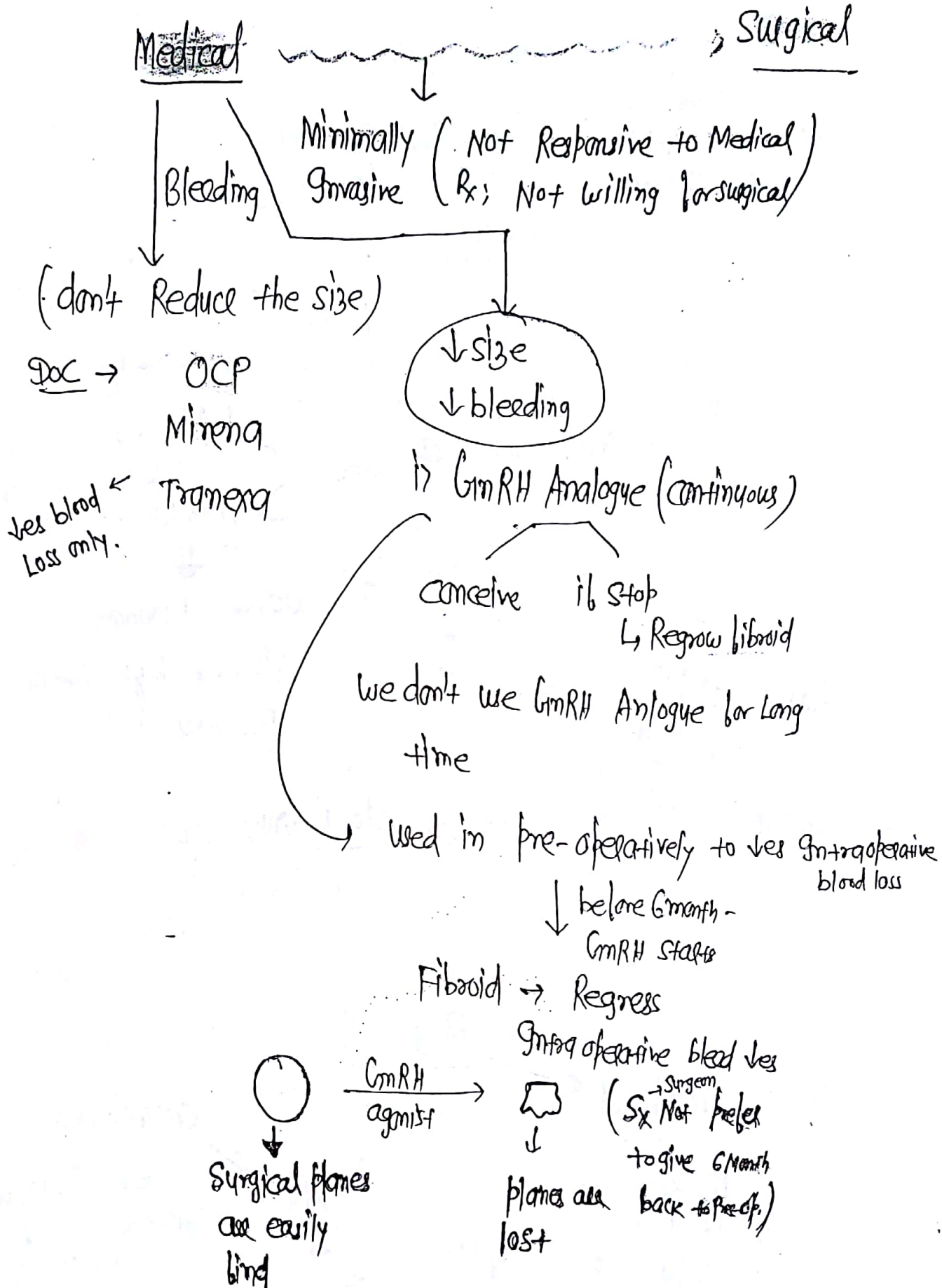
Subendometrial cyst

Poorly defined Junctional zone

On MRI  $\Rightarrow$  Junctional zone thickness  
 $> 12\text{mm} \Rightarrow$  Likely adenomyosis

$< 8\text{mm} \Rightarrow$  Unlikely "

# Tt for Fibroid $\Rightarrow$



Drugs that decrease size of Fibroid →

U → Ulipristal

Are → Aromatase Inhibitor

Gynae → GnRH Agonist/Antagonist

M → Mifepristone

D → Danazol

ii) GnRH Antagonist

iii) Mifepristone } Progesterone (R)

iv) Ulipristal } Modulator

(wants to conceive  
↳ don't give)

↳ Aromatase Inhibitor - Anastrozole

(S/E - Severe Hypoestrogenic)

• Minimally Invasive

UAE → Uterine artery embolization

Embolization

do if family is complete

MRg HIFU

Magnetic Resonance

Guided High Intensity

Focused US

• Rapid Symptom Improvement

• Fibroid keep shrinkage

do if only family is complete;  
expensive

\* Surgical O/T

Radical

Hysterectomy

M/C Indication for

TAH. → Fibroid \*\*\*

Conservative

Myolysis

↓

Cryo  
Laser

Submucosal

(Arterio, V)

Myomectomy

↓

hysteroscopic  
Laparoscopic  
minimally

} others



# \* Gn Laproscopic hysterectomy

(189)

↳ Slightly higher Recurrence Rate

↳ Not Statistically significant.

\* Laparoscopy is better than Abdominal in all except  $\Rightarrow$  Recurrence

\* M/c Side effect of Hyster.  $\Rightarrow$  Uterine Perforation  
scopy fluid overload

\* Myomectomy  $\Rightarrow$  It is enucleation of Myomata from the uterus leaving behind a potentially functioning organ capable of Future Reproduction

## INFECTIONS

Bacterial  
vaginosis

Candida

Trichomonas

- all causing vaginal discharge/vaginitis

- M/c - Bacterial vaginosis > Candida > Trichomonas

- Gardnerella

vaginalis

(Mycoplasma

Ureaplasma

Mobilicoccus)

Doderline Replaced  
by Gardnerella

Candida albicans

T. vaginalis

after passing urine; Pain left  
blc of excoriation of skin

- Foul Smelling discharge

itching (X)

Dyspareunia (X)

Urinary symptoms (X)

- Pruritis

- Discharge may present

- Urinary symptoms (X) (SPASH SYNDROME)

- Discharge

$\pm$  Dyspareunia

$\pm$  Urinary symptoms

$\pm$  Pruritis



Discharge  $\Rightarrow$  Foul Smelling  
Off white / Greyish

Curdy  
White

Green  
(Yellow)

pH  $> 4.5$

$< 4.5$

$> 4.5$

- IOC - Saline Microscopy

Clue cells (+)

Pseudohyphae

organism

- Ratio of Poly Morpho-Nuclear cells  
epithelial cells  
 $< 1$

See itself  
 $\downarrow$   
dit + flagella

- Gold standard - Gram Staining

Nugent Score  $\Rightarrow 7-10$

Culture has No Role in  
Bacterial vaginosis

Culture in  
SDA Media

Culture in  
Stuart's medium

- Amsel's Criteria

if 3 out of 4 - Bacterial  
vaginosis

Splash  
dysuria

Strawberry  
cervix

i) Foul Smelling (off/white)  
discharge

$\downarrow$   
Painful Urination  
due to exfoliation of vulva

(Punctate Hemorrhage)  
yes

ii) pH  $> 4.5$

Whiff test - (-)

Whiff test - (+)

iii) Clue cells (at least 20% of  
the epithelial cells)

iv) Whiff test (addition of KOH to D/S  $\rightarrow$  Fishy Amine odour)

Not a STD

Usually  
Not  
STD

STD

Partner Not  
treated

Usually Not  
(done if partner  
is symptomatic)

Yes

\*\*  
Doc  $\Rightarrow$  Metronidazole  
 $\downarrow$   
Clindamycin

Single dose  
clindamycin

Metronidazole

• Can cause Pre-term\*\*  
Labour in ♀

• Recurrent  
Vulvo vaginal candidiasis

$\downarrow$   
 $\geq 4$  episodes/year\*\*

PID (Pelvic Inflammatory Disease)

- Infection of upper genital tract  
(uterus | Fallopian tube | ovaries)

M/c organism  $\Rightarrow$  Chlamydia  
+  
Gonorrhea

age group  $\Rightarrow$  15-25yr

highest R/F  $\Rightarrow$  Multiple sexual partners.

PID in virgin girl  $\Rightarrow$  d/f Tuberculosis

PID in GUD users  $\Rightarrow$  d/f Actinomyces

\* Clinical diagnosis  $\Rightarrow$

$\hookrightarrow$  Pain Lower abdomen & Any of the following

a) Uterine tenderness

b) Cx Motion //

c) Adnexal //

Ix  $\Rightarrow$  \* USG  $\Rightarrow$  Cog wheel sign; Beads on string sign; Waist sign

Endometrial Bx  $\rightarrow$  Plasma cell endometritis



Chronic endometritis



IT of chlamydia

Laparoscopy  $\rightarrow$  Best (Gold standard)

$\hookrightarrow$  Fitz-Hugh Curtis Sy.

# FIB - Hugh Curtis Syndrome

↳ Pelvihepatitis

violin string Adhesions b/w Liver & Anterior  
Abdominal wall

Rt. upper Quadrant Pain

Liver enzyme (N)

M/c caused by ⇒ Chlamydia & Gonorrhoea

\* Long term complication of PID ⇒

i) Infertility;

ii) Ectopic ♀;

iii) Chronic Pelvic Pain;

iv) Recurrent PID;

v) Hydrosalpinx.



# CONTRACEPTION

\*

## Methods of contraception

### TEMPORARY METHODS

(Used to postpone pregnancy or space birth)



- BARRIER METHOD ;
- NATURAL CONTRACEPTION ;
- OCPs ;
- Injectables
- Implants
- GUCDs

### PERMANANT METHODS

(Aim is to Purposefully & Permanently destroy the Reproductive capacity of an individual).

Female



Tubectomy

Male



Vasectomy

### \* OCPs

\* on the basis of Amount of estrogen; they can be classified

as → i) Low dose Pills :  $< 50 \text{ mcg}$  (Avg:  $35 \text{ mcg}$ ) Ethinyl Estradiol

ii) High dose Pills :  $\geq 50 \text{ mcg}$  (Ethinyl Estradiol)

iii) Very Low dose Pills :  $\leq 20 \text{ mcg}$  (Ethinyl Estradiol)

iv) Lowest Possible Pills :  $10 \text{ mcg}$  (Ethinyl Estradiol)  
(~~Lo~~ 10 Estrogen)

### \* Mala D & Mala N

↳ both have  $30 \text{ mcg}$  Ethinyl Estradiol +  $0.15 \text{ mg}$  Levonorgestrel (LNG)  
↳ Both have 21 hormonal tablets & 7 ferrous fumarate tablets;

Mala D available @ a cost of 2 Rupees; while Mala N free of cost by govt of India.

M.O.A of OCP  $\rightarrow$  Mainly :

Inhibition of ovulation.

\* M/c Side effect of OCP :

Breakthrough bleeding

(192)

\* In Anovulatory SUB



Estrogen breakthrough bleeding

$\downarrow$   
Progesterone breakthrough bleeding.

\* If a Lady Misses 1 pill ; take 2 pills the following doses ;  
if she misses 2 pills  $\rightarrow$  Backup Method of contraception

\* In the event of Missing a pill :

Take the Most Recent missed pill immediately ; use  
condoms for 7 days & continue the packet

$\downarrow$   
Now if

$\swarrow$   
 $\geq 7$  pills are Remaining in packet

Finish the Remaining tablet &  
Start the New packet after 7 days  
gap.

$\searrow$   
 $< 7$  pills are Remaining in  
packet

Finish the Remaining pills  
& Start the New packet  
Next day without a 7 days  
gap.

Q: A women who is taking combined ocp misses 2 consecutive pills.  
There are 10 pills Remaining in the Packet. Next 7/11

a) Take both pills immediately continue the packet & use condom for 7 days

b) Take the Most Recent missed pill immediately ; continue the packet & use  
condom for 7 days.

c) Take the Most Recent missed pill immediately ; continue the packet ; use condom for  
7 days & commence the Next packet after a 7 days gap.

\* Fertility Return  $\Rightarrow$   $\tau$  (in 3 Months of withdrawal of the drugs in 90% cases)  
 $\downarrow$   
(Ovulation Return)

\* Which contraception have least chance of Ectopic  $\Rightarrow$  OCP

\* OCP also ↓ Risk of PID.

But  $\uparrow$  Risk of candida & chlamydia are seen by combined oral contraceptives

\* Other M.O.A. of OCPs  $\Rightarrow$  Prevention of Fertilization;  
Interference  $\tau$  Implantation;

\* OCP & Cancers  $\Rightarrow$

• OCP & Cervical cancer  $\rightarrow$   $\uparrow$ es;

• Ovarian cancer  $\downarrow$ es by 50%;

• Endometrial cancer  $\downarrow$ es by 60%;

• Colon cancer  $\downarrow$  risk;

• Breast cancer : No  $\uparrow$ es Risk

$\hookrightarrow$  OCPs are protective against benign breast disease;

but Role of OCPs are controversial in Ca of Breast,

• Using HRT (Hormone Replacement Therapy)  $\uparrow$ es chance of breast cancer.

• Hepatic Adenoma :  $\uparrow$ es Risk;

• HCC : No  $\uparrow$ es Risk (Not decrease Risk also);

• Gallbladder cancer : No  $\uparrow$ es Risk



\* Absolute c/I of OCP (WHO category 4)  $\Rightarrow$

(193)

Mnemonics

L

Banks  $\rightarrow$  K/c/o Breast Cancer

Have  $\rightarrow$  Severe Hypertriglyceridemia / Hypcholesterolemia

Various  $\rightarrow$  Undiagnosed vaginal bleeding

Scheme  $\rightarrow$  Stroke ; Smoker over age of 35 years.

To  $\rightarrow$  Thrombophlebitis | Thromboembolic disorder

Provide  $\rightarrow$  Pregnancy

Home  $\rightarrow$  Uncontrolled Hypertension ( $\geq \frac{160}{110}$  mmHg)

Looms  $\rightarrow$  Acute Liver disease (Hepatitis ; Cirrhosis / Not c/I of OCP)

During  $\rightarrow$  Diabetes  $\bar{c}$  Vasculopathy

May  $\rightarrow$  Migraine  $\bar{c}$  Aura (i.e Focal Neurological deficit).

also; common Artery Disease is absolute c/I of OCP

\* Newly Married couple : choice of contraception : OCP

Living far apart ; Meeting occasionally

L Barrier Method

\* Safety : Which contraception is safest : "Barrier"

$\downarrow$

S/E  $\Rightarrow$  very high failure Rate

$\therefore$  Pregnancy is S/E



## PROGESTERIN ONLY PILLS (POP)

aka "Minipills" (75mcg Progesterone)

M.O.A. : Thickening Cervical Mucus

\* Should be taken on Same-time Everyday



(Safe Margin < 3hrs)



If delay was for > 3hrs - Back Method should be used

\* POPs are contraception of choice in Lactating Female



↳ Can be started Immediately after delivery, POP > GUD

\* Contraception of choice in Lactating's Female : POP > GUD

↳ but Not Lactating Amenorrhoea

↳ b/c high failure Rate

\* Minipill available in India ⇒ "CERAZETTE"



M.O.A. ⇒ Mainly Again

↳ Inhibition of ovulation

Safe Margin : 12 hrs  
(A delay of ~ 12 hrs can be accepted)

\* M/c side effect of POP : Irregular vaginal bleeding.

\* Absolute C/I :

- i) Pregnancy;

- ii) Undiagnosed vaginal bleeding;

- iii) K/c/o Breast Cancer

} Same for all

## PROGESTERONE INJECTION

DMPA (Depot Medroxyprogesterone  
Acetate)

Dose  $\Rightarrow$  150 mg i/m

To be Repeated every 3 Months

Pt. can wait upto 4 weeks late  
for Next Injection

385  
(194)  
Net En (Norethisterone  
enantate)

200 mg i/m

Repeated every 2 Months

Pt. can wait upto 2 weeks  
Late

\* DMPA  $\Rightarrow$  2 Good things  $\Rightarrow$  i) Useful in Female  $\bar{c}$  Epilepsy  
 $\uparrow$  Seizure threshold;  
ii) Reducing Sickling crisis: Useful in Sickle cell Anemia;

2 Bad things  $\Rightarrow$  i) Significant Bone loss;  
ii) Delay in Return of Fertility;  
Avg. delay: 12 Months  
Max<sup>m</sup>. delay: 18 Months.

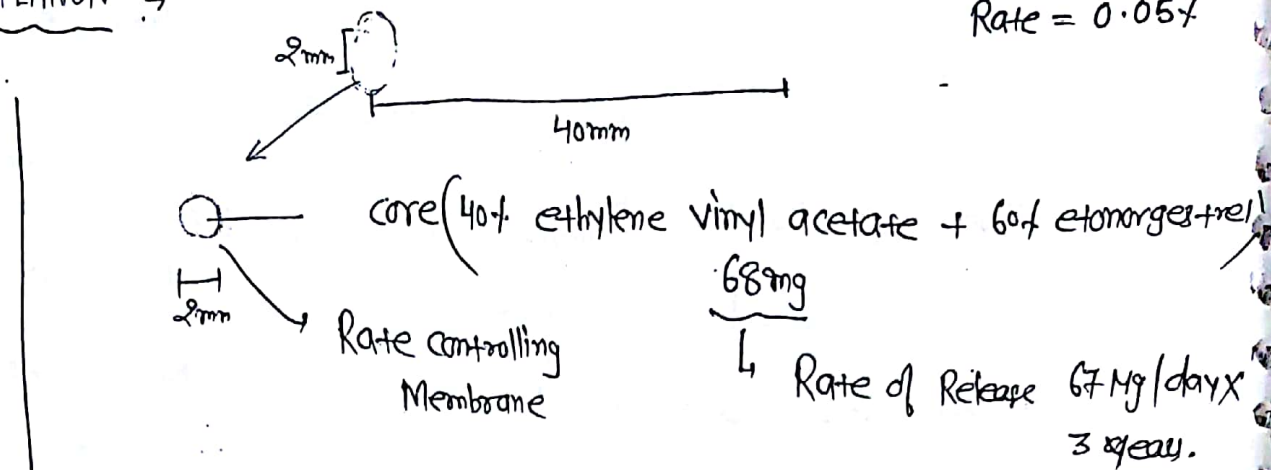
\* M/c side effect of DMPA: Irregular Vaginal Bleeding.

\* Absolute C/I: Same three S/E

↳ Undiagnosed vaginal bleeding  
Pregnancy  
K/c/o Breast cancer

PROGESTIN ONLY IMPLANTS  $\Rightarrow$  Among all contraceptives; Least failure Rate = 0.05%

IMPLANON  $\Rightarrow$



Single Rod  $\bar{c}$  Keto-desogestrel

OPD Procedures (Most Popular Implant Nowadays);

M.O.A.  $\Rightarrow$  Inhibition of ovulation.

Site of Implantation  $\Rightarrow$  Non-dominant Arm (Medial aspect of Upper Arm).

It is Not Radioopaque.

Next Generation of IMPLANON: NEXPLANON  $\rightarrow$  Radioopaque

M/c side effect: Irregular vaginal bleeding.

Absolute C/I:

Same 3

- i) Pregnancy
- ii) Undiagnosed vaginal bleeding
- iii) K/c/o Breast cancer

NUVA

It is Vaginal Ring (Blue or white);

It is E+P Compound  $\left( \begin{array}{l} E = \text{Ethinyl Estradiol} : 15\mu\text{g/day} \\ P = \text{Etonogestrel} : 120\mu\text{g/day} \end{array} \right)$

M.O.A.: Inhibition of ovulation.



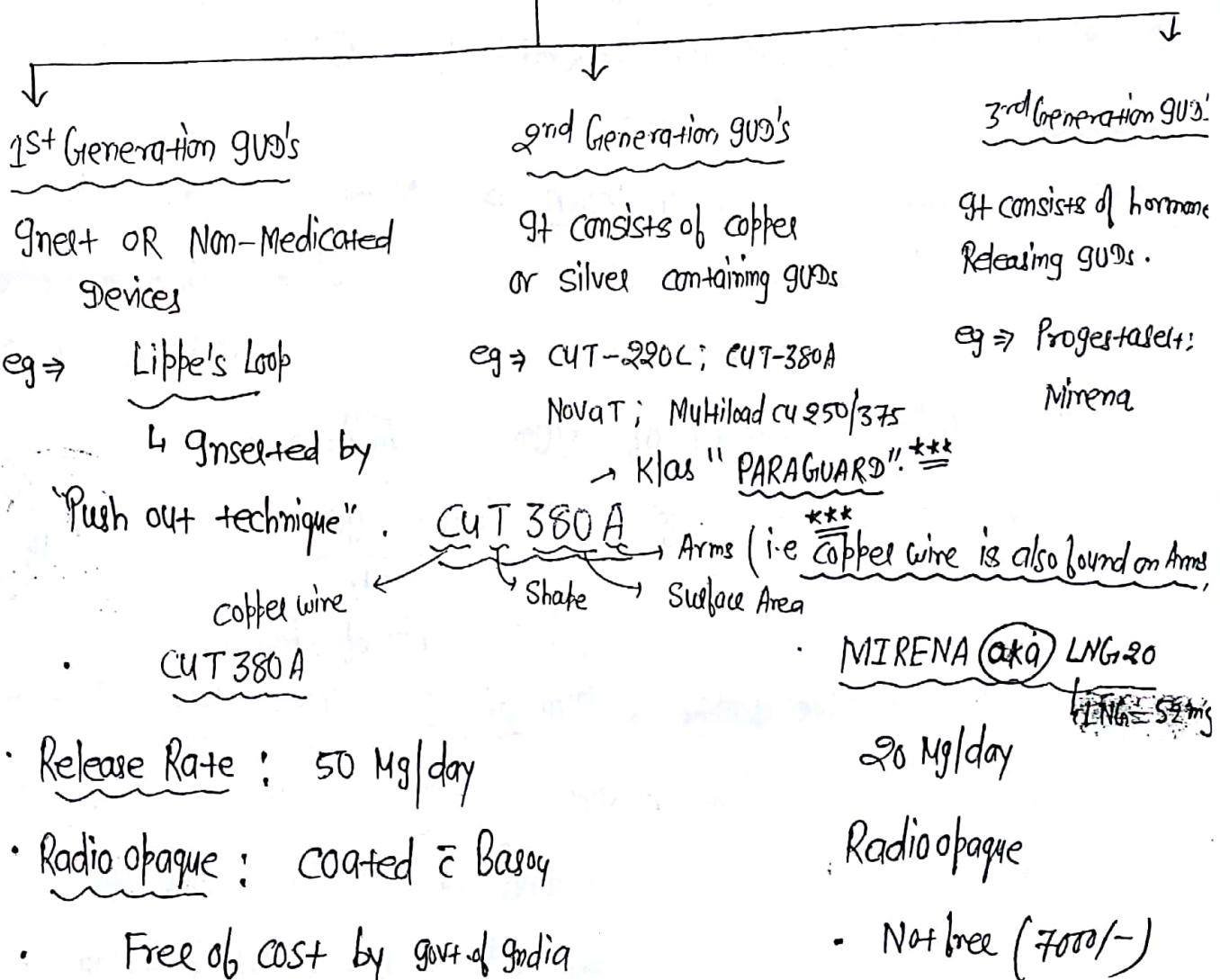
How to Use : Insert in vagina on 1<sup>st</sup> day of <sup>387</sup> menstrual cycle & keep it for 3 weeks;  
after 3 week; Last week is "Ring free" (195)

↓  
then: Insert New Ring.

Safe period of Ring : 3 hours before sex.

It means if the expelled Nuva Ring has been out of vagina for more than 3 continuous hours; during weeks 1 & 2; you may not be protected from @.

## INTRA UTERINE DEVICES





## CUT 380A

- Effective for span of 10 years;
- presence of beak : to less Risk of  
Perforation & for Identification  
and Removal.
- Can be used as Emergency contraceptives
- Less Menstrual blood Loss

\* M/c Side effect of GUD  $\Rightarrow$   $\uparrow$  Bleeding;  
M/c cause for Removal  $\Rightarrow$  Pain  
of GUD

\* G.U.D. : Mech<sup>m</sup> of Action  $\Rightarrow$  Mainly "Spermicidal"  
 $\therefore$  Ans  $\rightarrow$  Inhibition of Fertilization  
Inhibition of Implantation

M/c Infection of GUD : Actinomyces;

\* GUD insert  $\bar{c}$   $\odot$  : Remove GUD  
 $\downarrow$  why  
b/c  $\uparrow$  Risk of Abortion

\* \*\* Do USG b/c failure of GUD  $\odot$   $\bar{c}$  G.U.D. Mostly; Ectopic \*

\* Max<sup>m</sup> Infection  $\bar{c}$  GUD :  $\bar{c}$  / in 20 days of Insertion \*

\* Multifilamentous : Older days  $\rightarrow$   $\uparrow$  Risk of Infection;

\* Monofilamentous : Now a days  $\rightarrow$   $\downarrow$  Risk of Infection  $\rightarrow$  that too by design

## Mirena

- Effective for span of 5 years.
- Drug presents on lower  
limb only,  
(drugs Release for a span  
of 7 years).
- Not an Emergency  
contraceptives
- Less Menstrual blood loss  
(amenorrhic for at least  
6 Months).

Misplaced IUD (Missing thread)  $\Rightarrow$  G.O.C  $\Rightarrow$  USG <sup>389</sup>  
 $\downarrow$  if Not found  
 X-Ray <sup>(196)</sup>

- \* IUD Missing  $\bar{c}$  Perforation  $\oplus$  in Abdomen: Mx: Laparoscopy\*\*
- \* if IUD Embedded in Myometrium: Mx: Hysteroscopy & Remove it.

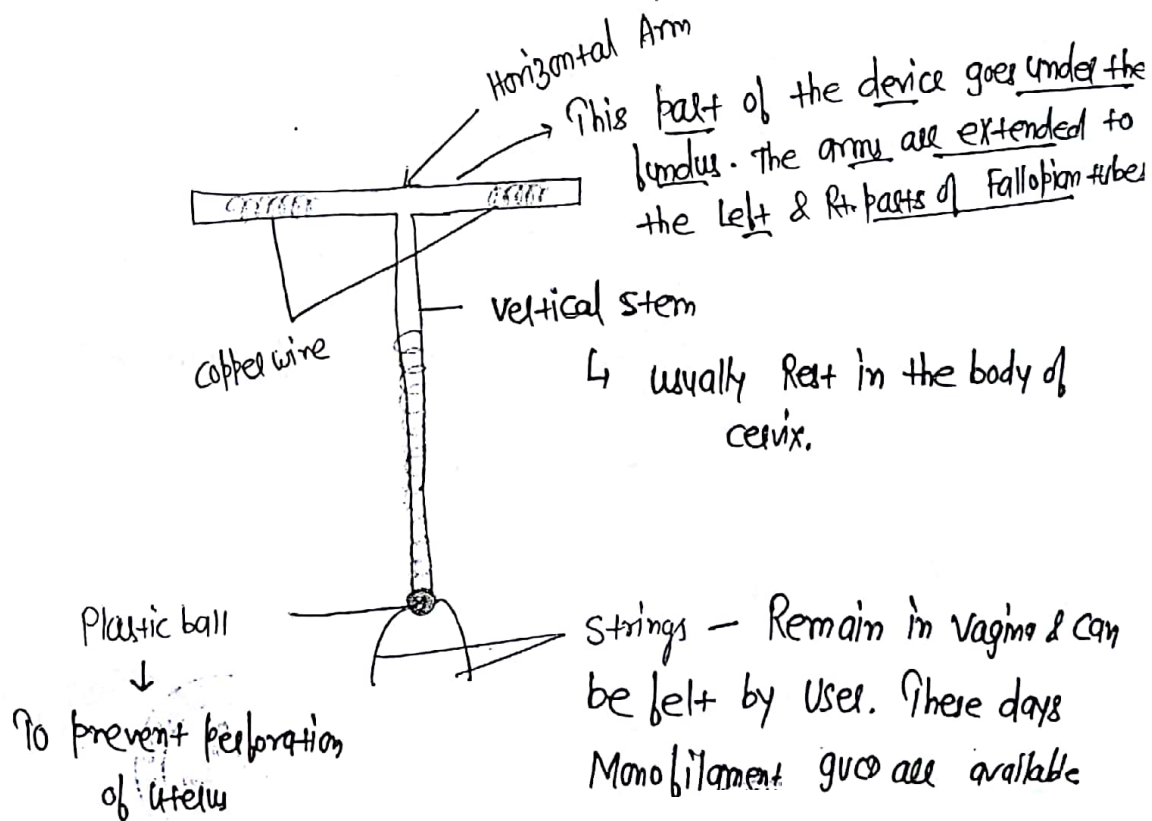
\* Contraception in HIV $\oplus$  Patient  $\Rightarrow$  IUD + Barrier  
 D.M.  $\Rightarrow$  IUD  
 Heart disease  $\Rightarrow$  IUD

\* M/c Mode of Contraception Used in India: Barrier Method\*

\* Best Contraception; if Family is complete: Vasectomy > Sterilization > IUD <sup>ACP</sup>

Best Non-permanent Contraception; if Family is complete: IUD > ACP \*

\* No Risk of Teratogenicity; if a  $\oplus$  Female continue  $\oplus$   $\bar{c}$  IUD.



Cu-T 380A

Silver Line Cu 380 Ag  $\Rightarrow$  M/c Cu  $\pi$  used worldwide

$\hookrightarrow$  have flexible Arms: Silver core  $\oplus$  & Rest Same as CuT 380A

\* Absolute c/I of GUCD - category 4 of WHO ⇒

Mnemonic ⇒

- Please → Puerperal sepsis or Postabortal sepsis
- Don't → DUB / Unexplained vaginal bleeding
- Try to → Gestational Trophoblastic Disease
- Put → Current PID / STD or E/in Past 3 months
- Condom → Puerperal sepsis, know Pelvic TB  
Ca Cervix  
Ca endometrium



## STERILIZATION (Permanent Method) <sup>391</sup>

\* No. of children Required for sterilization? (197)

Ans  $\Rightarrow$  At least 1 child of 1yr old.

\* It is a legal procedure  $\rightarrow$  consent is Mandatory; but only of Client.

\* consent of spouse is Not Mandatory.

• 22yr - 45yr can undergo sterilization;

Q. Q. Unmarried women can undergo sterilization?  $\Rightarrow$  NO  
Married or ever married  $\Rightarrow$  Yes

• Most cost effective mode of contraception: Vasectomy

• Most effective contraception: Implant

• How many days after vasectomy should avoid:  $> 3$  Months till coitus or use another contraception azoospermia

• Postpartum Sterilization: Within 7 days of delivery @  
after 42 days of postpartum (can't do @ 8th or 9th days;

• This is klas "Interval Sterilization."

• M/C Method used for postpartum Sterilization: Minilaprotomy (3cm Long)

$\hookrightarrow$  by Pomeroy technique

$\Downarrow$   
Babcock forceps

• We don't use Laparotomy / Laproscopic Sterilization in Post-partum Sterilization.

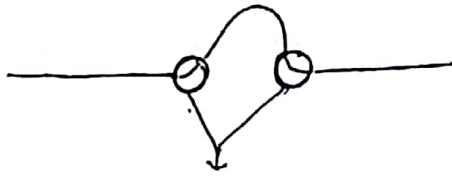
\* Post-placental GUD  $\Rightarrow$  if we put GUD in 10 min of delivery;

\* Post-partum GUD  $\Rightarrow$  if we put GUD in 48 hr of delivery,



- \* M/c part of Fallopian tube that we ligate in sterilization  
↳ Gsthmus.

Modified Pomeroy  
aka "Parkland Method"



double ligation of tube is done

Failure Rate = 0.2

Pomeroy



Middle part of tube (3-4 cm from fundus) is formed into loop using baby's loops, which is tied & excised. 0.4

- \* Interval sterilization → Non-pregnant state

Whenever we do sterilization in Non-♀ Female is k/as "Interval"

M/c Method used: Laparoscopy

- \* M/c Method of Female Sterilisation ⇒ Laparoscopic Sterilisation

CO<sub>2</sub> gas used

↓  
Intraabdominal Pressure b/w 8-12 mm ; Max<sup>m</sup> 2 15 mm of Hg

No + More than 15 mm of Hg b/c it does venous return

- \* M/c Method for Laparoscopic Ligation ⇒ Yarn balope Ring / Silastic band
- \* Site of Ligation ⇒ Gsthmus

- \* Among Sterilization technique ⇒

Least failure Rate : Unipolar Cautery > Modified Pomeroy  
(Never Used)  
↳ b/c of Intestinal burn

Highest Failure Rate : Clips > Bipolar cautery  
↓  
HULKA CLIPS

Reversal Methods : Clips > Falope Ring > Modified Pomeroy <sup>393</sup> > Cautery  
(198)

Laparoscopic Ligation → P+ in Lithotomy position

↓  
With help of Verres Needle (Introduced at an Angle of 45°) Pneumoperitoneum is created

↓  
CO<sub>2</sub> gas used  
Procedure is done under Sedation & Local Anesthesia

\* M/c used Method in Minilaparotomy : Modified Pomeroy

\* Highest Risk of ectopic @ : Cautery > Madlena > Modified Pomeroy

